TRANSACTIONS

AMERICAN ACADEMY
of
OPHTHALMOLOGY
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SEPTEMBER - OCTOBER
1956

Volume 60 Number 5

ANNUAL MEETING, OCTOBER 13-18, 1957, CHICAGO

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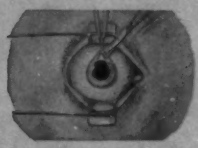

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American Academy of Ophthalmology and Otolaryngology

VOLUME 60 SEPTEMBER-OCTOBER, 1956 NUMBER 5

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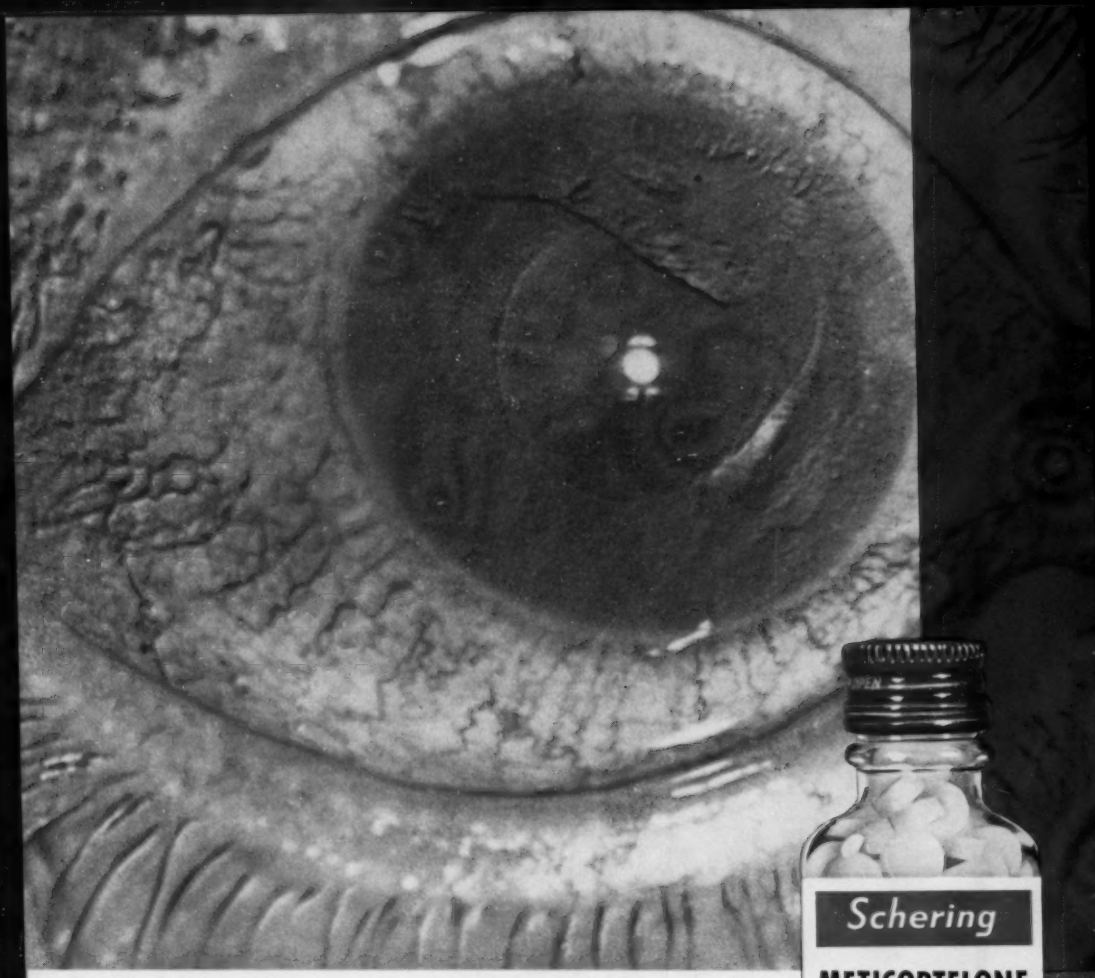

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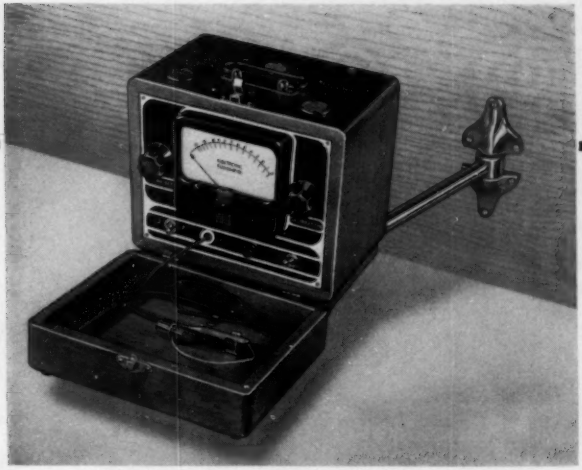

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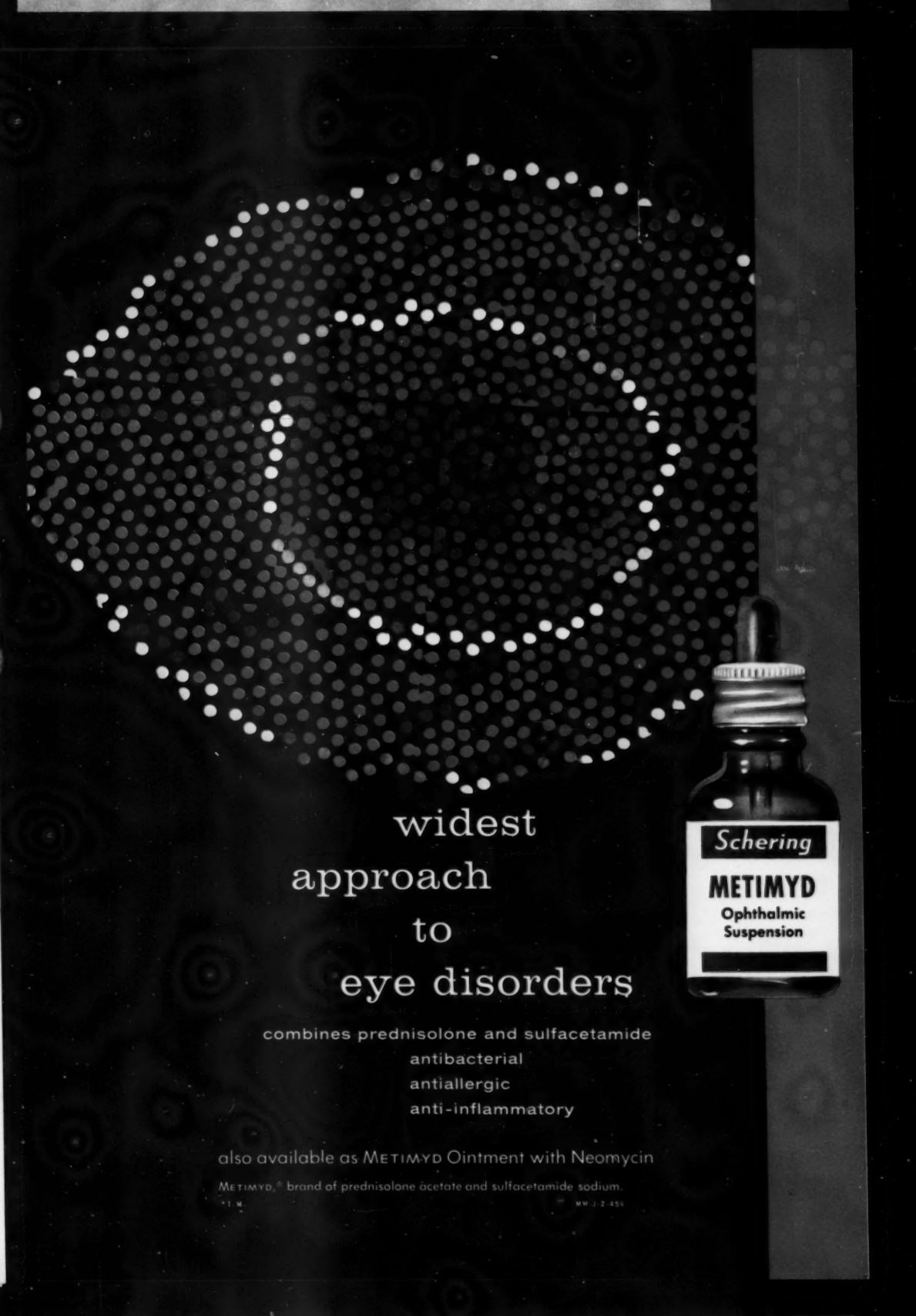
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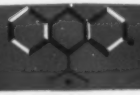

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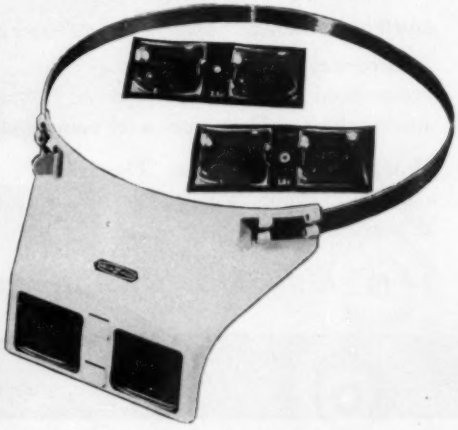

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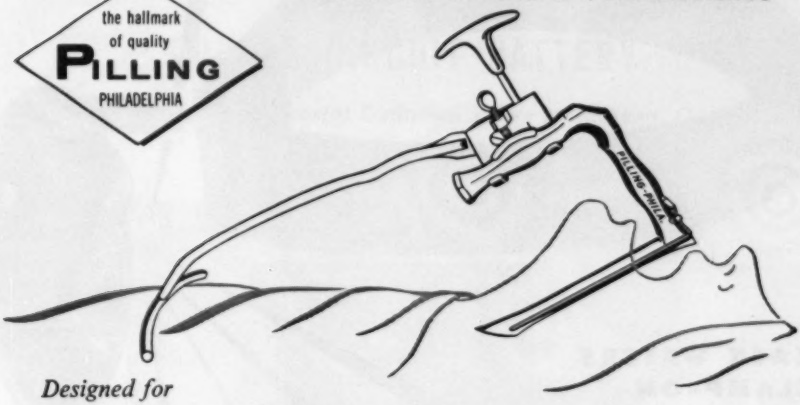

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Reference—R. B. Lewy: Acta Oto-Laryngologica, Vol. 46, Fasc. 1, 1956

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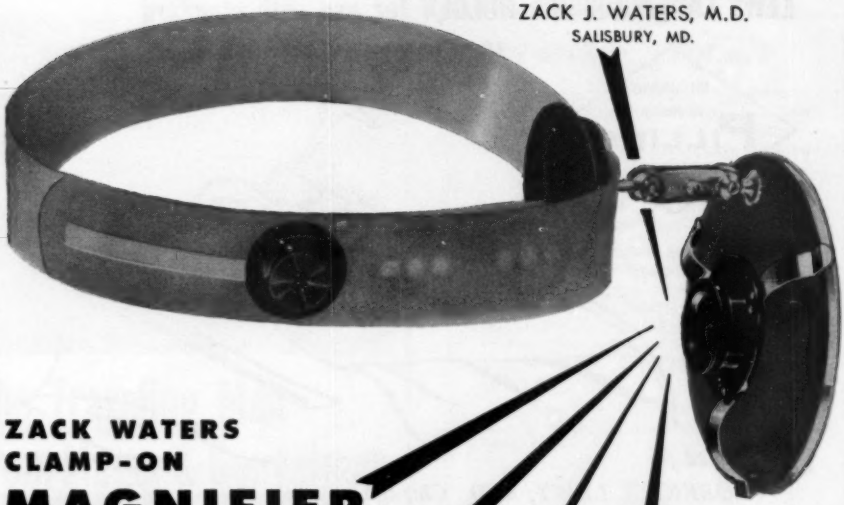

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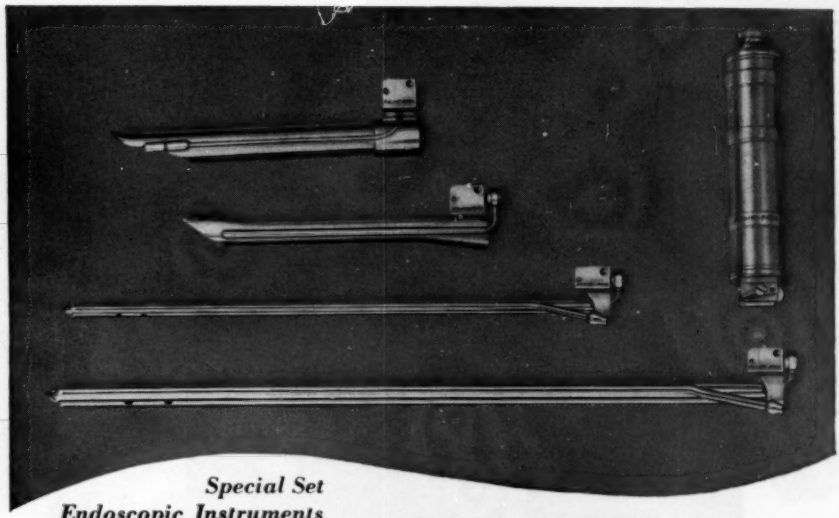

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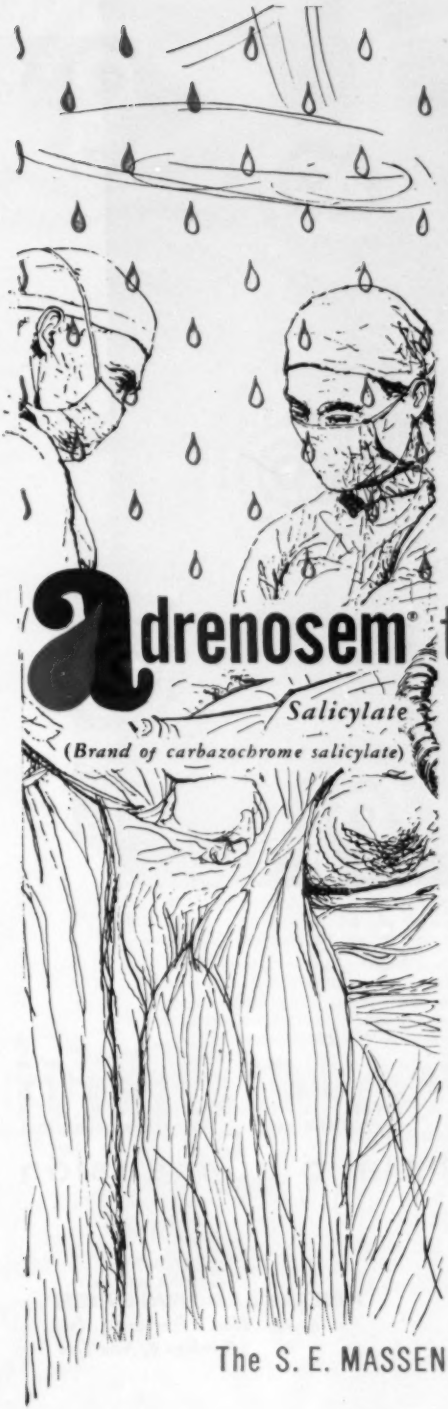

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1. *Bacala, J.C.: The Use of the Systemic Hemostat Carbazochrome Salicylate, West. J. Surg. 64:88 (1956).*

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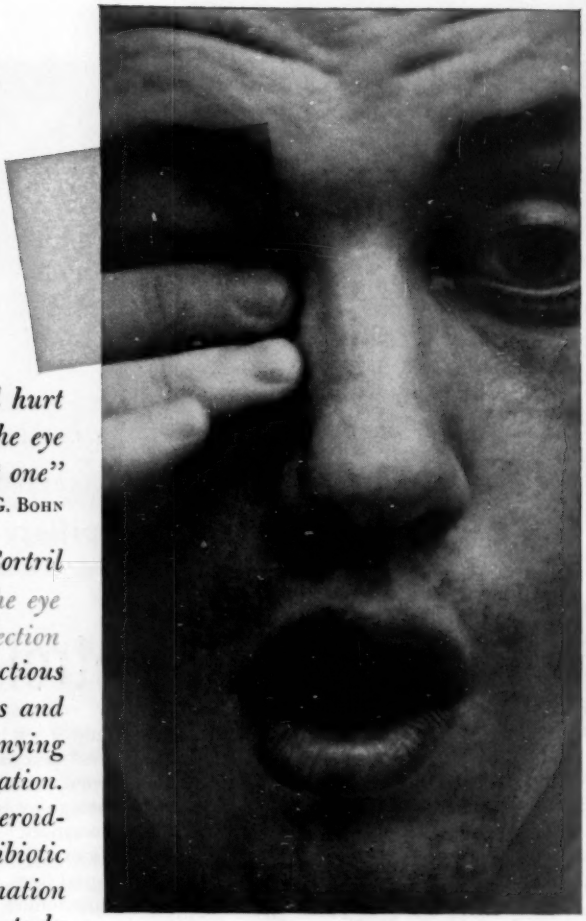

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1. Robinson, H. M., Jr., et al.: M. Times 83:227, 1955.

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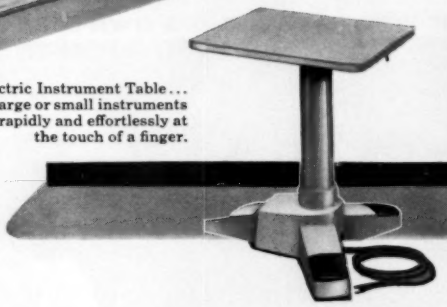

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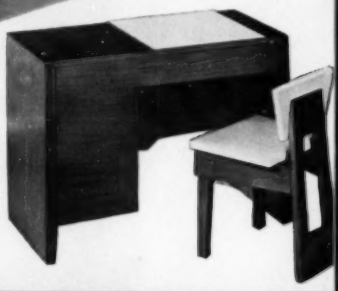

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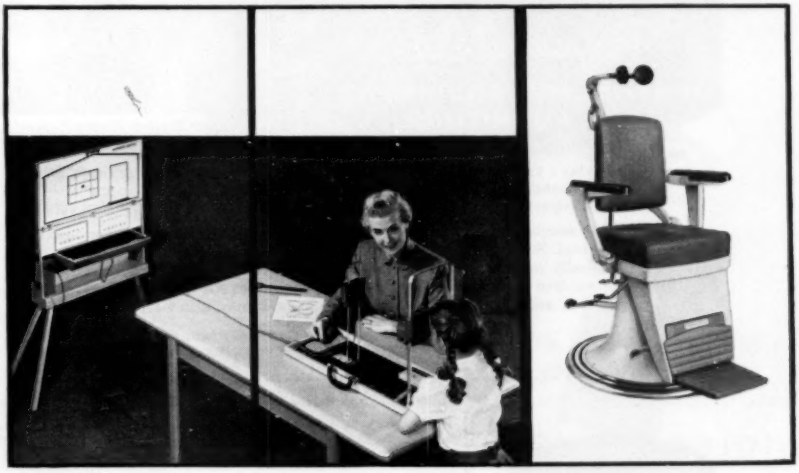

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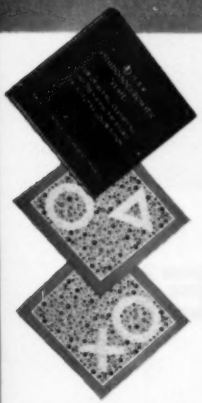

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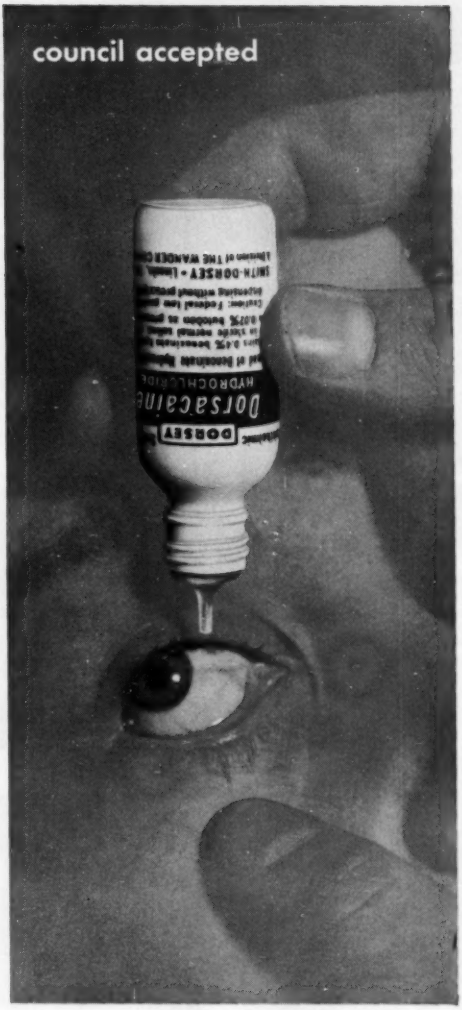

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*Schlegel, H. E., Jr., and Swan, K. C.: A. M. A. Arch. Ophth. *51*:663 (May) 1954.

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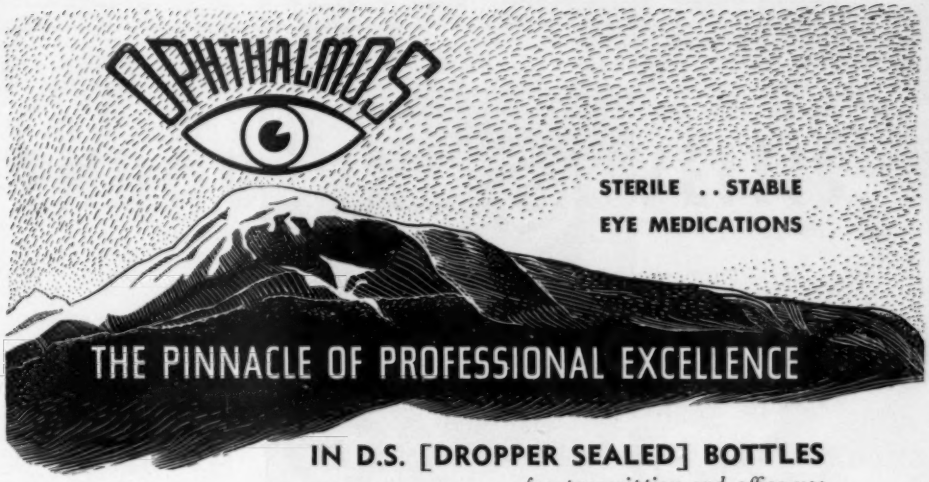

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1. Rasgorshek, R. H., and McIntire, W. C.: Am. J. Ophth. *40*:34 (July) 1955.
2. Council on Pharmacy and Chemistry, A.M.A.: New and Nonofficial Remedies, Philadelphia, J. B. Lippincott Company, 1955, p. 263.
3. Gettes, B. C.: A.M.A. Arch. Ophth. *51*:467 (April) 1954.
4. Gordon, D. M., and Ehrenberg, M. H.: Am. J. Ophth. *38*:831 (Dec.) 1954 (a review of 8 studies covering 1035 patients).

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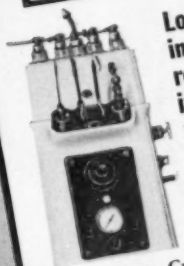

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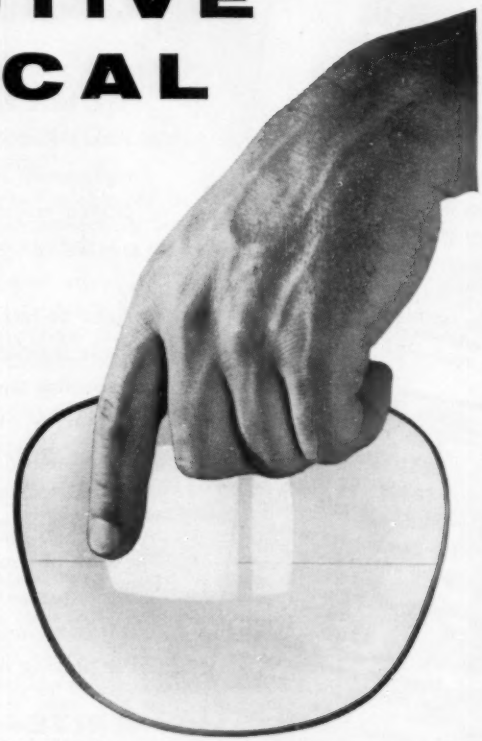

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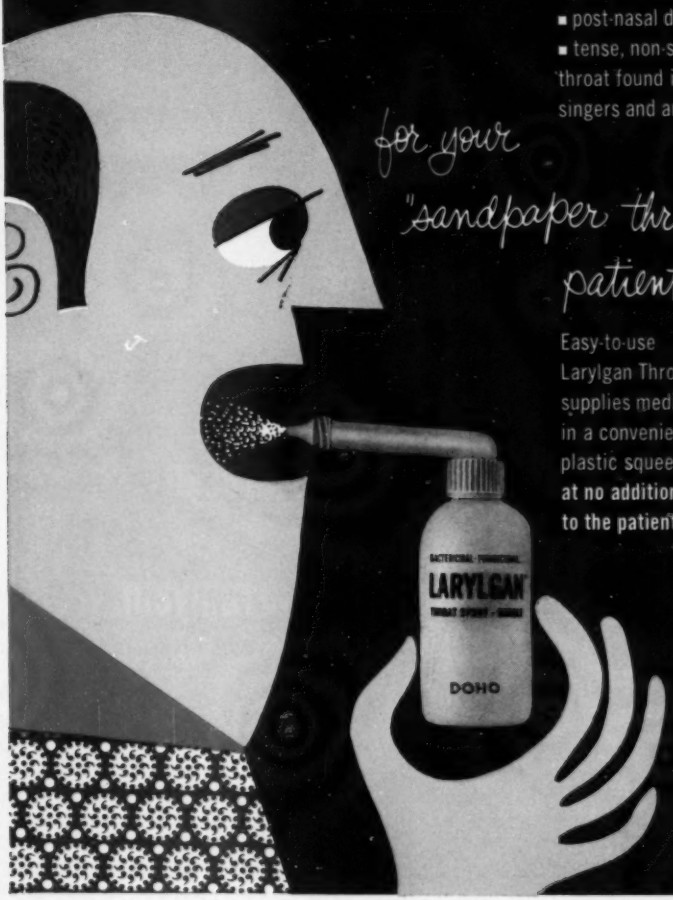

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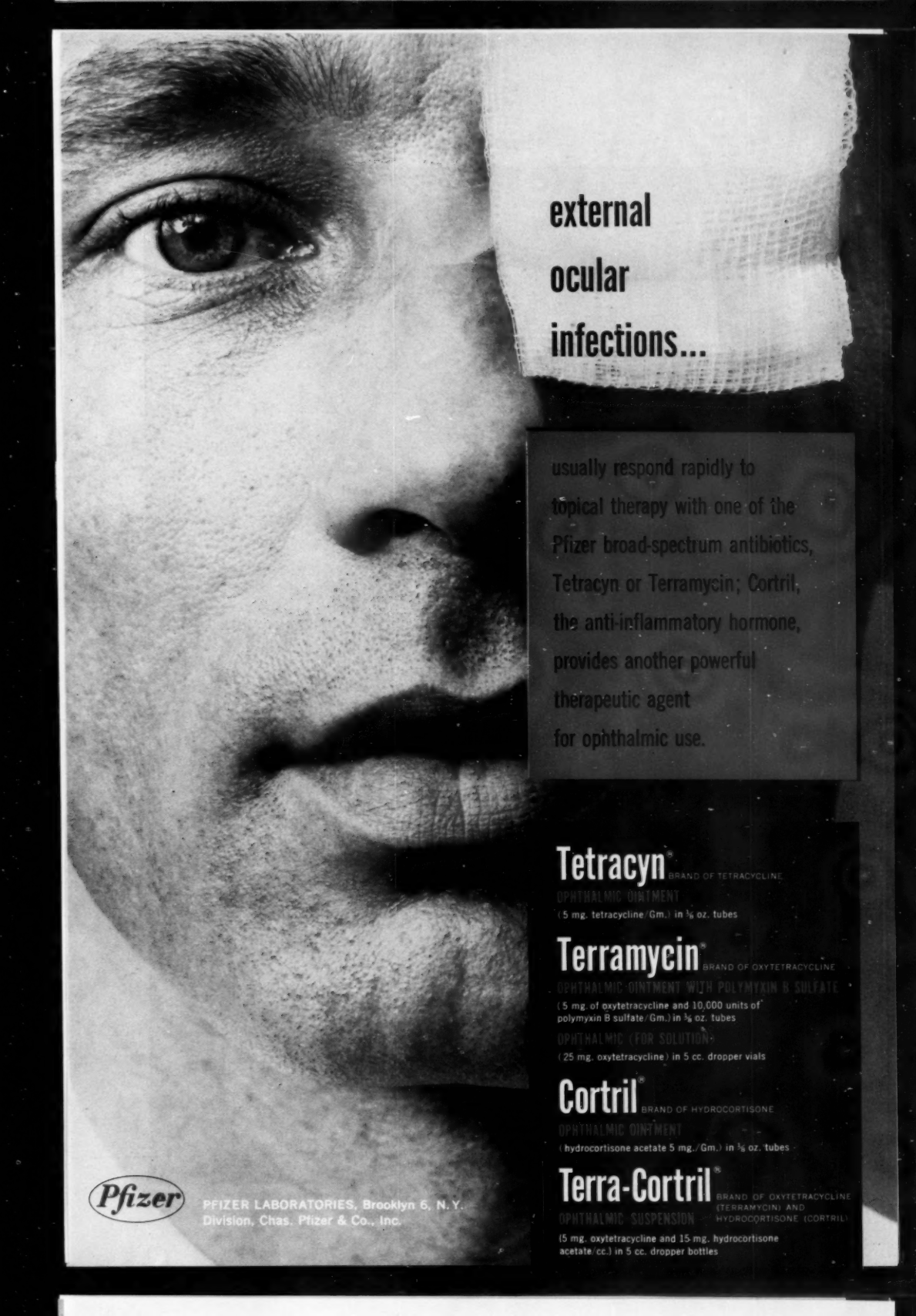
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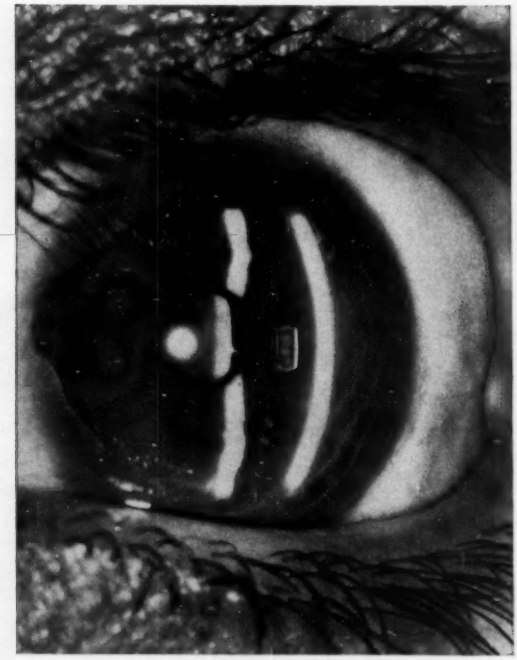

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Reference: Hogan, M. J., Thygeson, P. and Kimuras, J., Arch. Ophth. **53**:165, (Feb.) 1955.

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A COMPARISON OF THE TOXOPLASMIN SKIN TESTS, THE SABIN-FELDMAN DYE TESTS, AND THE COMPLEMENT FIXATION TESTS FOR TOXOPLASMOSIS IN VARIOUS FORMS OF UVEITIS

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IN October 1953, in a communication from the Wilmer Institute and the National Microbiological Institute, the results of the Sabin-Feldman dye test for toxoplasmosis on 409 stored serums from various types of patients were reported.[13] Ninety-eight of these serums were from patients without uveitis, 110 were from patients with nongranulomatous uveitis, and 201 serums were from patients with granulomatous uveitis. The various uveitis patients had been carefully studied in the Wilmer Institute to determine, if possible, the causes of their uveitis, and their histories were available for analysis. The serums of patients either without uveitis or with nongranulomatous uveitis were used as controls. As a result of this study, subject to possible fallacies in the dye test, it was concluded that adult ocular toxoplasmosis was an actual clinical entity and that Toxoplasma was the factor responsible for the ocular disease in approximately 25 per cent of patients with granulomatous uveitis. It was further pointed out that a diagnosis of toxoplasmic chorioretinitis could be made only by correlation of the ocular picture, the results of a careful medical survey to exclude other possible causes, and the results of serologic tests for toxoplasmosis. At that time there were an insufficient number of cutaneous tests to evaluate results of the toxoplasmin skin test. The most frequent ocular lesion in the patients believed to have toxoplasmosis was a focal chorioretinitis. There were an appreciable number of cases of generalized uveitis and a few cases of retinal periphlebitis.

Since the publication of this paper, there have appeared a number of important reports on this subject of ocular toxoplasmosis.[3,5,6,7,9,10,11] *Toxoplasma gondii* has been demonstrated in the enucleated eyes of patients with chorioretinitis and uveitis, and the results of

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the skin and dye tests on these patients are known. As a result of these and various other studies, it is now generally conceded that acquired toxoplasmosis is an important cause of adult granulomatous uveal disease, and that the skin and dye tests are usually positive in adult patients. However, while the evidence for these generalities is conclusive, nevertheless, in the individual clinical case, it is extremely difficult to evaluate the significance of the positive cutaneous and serologic tests, and there is no generally accepted criterion for a firm clinical diagnosis of acquired adult ocular toxoplasmosis. The difficulties arise from the relatively high incidence of positive results in apparently normal healthy individuals, the possible nonspecificity of the dye test,*[1] the improbability of isolating the parasite from the blood or extraocular tissues, and the absence of any associated systemic symptoms of infection with the parasite.

In the hope of resolving some of these difficulties, an analysis of the results of the toxoplasmin skin tests and of the serologic tests for toxoplasmosis has been made in 193 patients with uveitis studied in the Wilmer Institute between July 15, 1953, and July 1, 1955. In all these patients, a diagnosis of either nongranulomatous or granulomatous uveitis was made on the first ophthalmologic examination. Patients in whom such a diagnosis could not be made are not included in this study. Eighty-six patients were thus diagnosed as having nongranulomatous uveitis believed due to an allergic insult, and 107 were diagnosed as having granulomatous uveitis believed to be due to an infection of the uveal tract with some pathogenic agent causing granulomatous disease. Many of the patients with nongranulomatous uveitis were undergoing a period of remission between attacks, and the eyes were quiet. Practically all patients with granulomatous uveitis were in the active stages of the ocular disease. There was no particular difference in the age distribution of the patients with nongranulomatous uveitis and of those with various types of granulomatous disease (table I). Although the residences of these 193 patients showed a wide geographic distribution, the majority of them lived somewhere on the Atlantic seaboard.

All patients were subjected to a careful and extensive medical study to determine, if possible, the specific cause of the ocular disease. An attempt was made to ascertain, in the case of nongranulomatous uveitis, the specific bacterial hypersensitivity responsible for the allergic reaction; and in the case of granulomatous uveitis, any evidence of systemic disease which could cause the ocular granulomatous inflammation. The details of this medical survey have been outlined elsewhere.[12] Included in this survey were intracutaneous tests with toxoplasmin, and Sabin-Feldman dye tests on the serums. In about 50 per cent of the serums, complement fixation reactions against a toxoplasma antigen prepared from mouse peritoneal exudates were recorded.* In the performance of these tests, to avoid anamnestic reactions, the usual procedure was first to draw the blood for the serologic tests and later to do the toxoplasmin skin tests. The serologic studies were all made in the National Institute of Allergy and Infectious Diseases, and the skin tests and clinical studies were done in the Wilmer Institute. The intradermal antigen was prepared from mouse peritoneal exudates at the National Institute of Al-

*In the experience of one of us (L. J.), no confirmation of the reported cross-reactions has been obtained.[8]

*The complement fixation tests were performed by the Immunology Subsection, Laboratory of Tropical Diseases, NIAID, NIH.

TABLE I

AGE DISTRIBUTION IN YEARS OF PATIENTS WITH UVEITIS

TYPE OF UVEITIS	TOTAL NO. OF CASES	1-9	10-19	20-29	30-39	40-49	50-59	60 OR MORE
Nongranulomatous	86	3 (4%)	10 (11%)	13 (15%)	22 (26%)	17 (20%)	16 (18%)	5 (6%)
Granulomatous of conventional or unknown causation	58	0	5 (9%)	12 (21%)	14 (24%)	12 (21%)	9 (15%)	6 (10%)
Granulomatous—toxoplasmic origin	37	0	6 (16%)	7 (19%)	16 (43%)	7 (19%)	1 (3%)	0
Granulomatous—due either to toxoplasmosis or to conventional causes	12	0	0	6 (50%)	3 (26%)	1 (8%)	1 (8%)	1 (8%)

lergy and Infectious Diseases following the method of Frenkel.[4] The skin tests were read on a 0 to 4-plus scale. A one-plus reaction indicates erythema of 5 to 7 mm. with no, or only slight, induration; a 2-plus reaction, erythema of 8 to 15 mm. with some induration; a 3-plus reaction, erythema of 15 to 25 mm. with definite induration but without necrosis; and a 4-plus reaction, an equally extensive erythema and induration and also some evidence of necrosis. The positive reactions were all of the delayed type, coming on 24 to 48 hours after the test injection.

In a preliminary review of the histories of these 193 patients, it was immediately apparent that the results of the dye tests in the nongranulomatous group of patients were almost exactly the same as those found in the control cases of the first series. Since there was no question of a possible toxoplasmic causation in the patients with uncomplicated nongranulomatous uveitis, the results of these diagnostic tests for toxoplasmosis in the group with nongranulomatous uveitis were again used as controls for the evaluation of the results of these same tests in the group with granulomatous uveitis.

When the case histories of the patients with granulomatous uveitis were reviewed in the light of the results of the medical survey and of our former ideas of the proper interpretation of the diagnostic tests for toxoplasmosis, it was found they could readily be divided into four general groups. These were (a) patients in whom the uveitis appeared clearly attributable to one of the conventional causes; (b) patients in whom tests showing positive indications of toxoplasmosis offered the only evidence for an etiologic diagnosis; (c) patients in whom there was evidence of a conventional explanation of the uveitis, and also evidence of Toxoplasma infection, thus raising the question of alternative diagnoses; and (d) patients in whom the diagnostic survey had not revealed sufficient evidence of any granulomatous disease to justify an etiologic diagnosis.

When the case histories of the patients in whom positive tests for toxoplasmosis offered the only explanation of the uveitis were further reviewed, it was found that they all fulfilled each of the following criteria: (1) They showed an ocular picture believed to be compatible with ocular toxoplasmosis, i.e., a focal chorioretinitis, old glial scars with a super-

imposed acute allergic reaction, a generalized granulomatous uveitis, or (in the first series) a retinal periphlebitis. (2) All patients reacted positively to the skin test for toxoplasmosis. In all patients except one, results of dye tests were positive in a minimum titer of 1:64. The exception was a child, 10 years old, who had a nongranulomatous uveitis superimposed on old glial scars. In this patient, the skin test was 3-plus and the dye test was 1:16. (3) In none of these patients was there any evidence of other granulomatous disease to which the uveitis could logically be attributed.

These three criteria were then adopted as the requisites for a diagnosis of ocular toxoplasmosis in the individual case, and the case histories of the patients with granulomatous uveitis were again reviewed. On the basis of the final etiologic diagnoses the cases were classified into the four general groups which had been established: (a) conventional, (b) toxoplasmic, (c) uncertain, and (d) undetermined. The results of the various diagnostic tests for toxoplasmosis in the control group and in these various types of granulomatous uveitis are given below.

RESULTS

Incidence of Positive Skin and Dye Tests

The results of the cutaneous tests with toxoplasmin and the dye tests on the serums of the patients with the two types of uveitis are shown in table II. Thus, in nongranulomatous uveitis the total incidence of positive skin tests was 30 per cent and of positive dye tests was 20 per cent. Both tests were positive in 14 per cent of these cases. In contrast, in the granulomatous uveitis group, 61 per cent of the patients showed positive skin tests, 62 per cent showed positive dye tests, and both tests were positive in 54 per cent. These figures are quite comparable to the results previously reported in the first series of stored serums from uveitis patients.

Etiologic Diagnosis

The number of patients in each etiologic group of granulomatous uveitis and probable incidence in each are shown in table III. For comparison, the results of the etiologic study on the patients in the first series are also shown. It must, however, be remembered that in the first series, the diagnoses of ocular toxoplasmosis were all tentative and were based on the delayed results found in the stored serums and on the previous medical surveys.

In the group of cases due to conventional causes, there were 29 patients, an incidence of 27 per cent, as compared to an incidence of 42 per cent in the first series. This drop is undoubtedly partly due to the introduction of the therapeutic trial test for ocular tuberculosis and partly to stricter diagnostic criteria adopted for the diagnosis of other granulomatous disease entities as the etiologic factors. For only one patient in this group were results of both skin and dye tests for toxoplasmosis positive. In this patient, the reaction to the skin test was 2-plus and the results of the dye test positive in a titer of 1:64. Despite these positive findings, the other evidence in favor of a tuberculous causation was considered more convincing.

The number of cases ascribed to adult ocular toxoplasmosis was 37, an incidence of 35 per cent. The reason for this increase is not apparent.

The incidence of uveitis of uncertain causation dropped from 18 per cent in the first series to 11 per cent in this series. This may well be due to the stricter criteria mentioned above. These same factors would also account for the in-

TABLE II

RESULTS OF SKIN AND DYE TESTS IN DIFFERENT TYPES OF UVEITIS

TYPE OF UVEITIS	TOTAL NO. CASES	POSITIVE SKIN TESTS		POSITIVE DYE TESTS		POSITIVE SKIN AND DYE TESTS	
		NO.	PERCENTAGE	NO.	PERCENTAGE	NO.	PERCENTAGE
Nongranulomatous	86	26	30	17	20	10	14
Granulomatous	107	65	61	67	62	58	54

TABLE III

PROBABLE INCIDENCE OF ADULT TOXOPLASMOSIS IN CAUSATION OF GRANULOMATOUS UVEITIS

MATERIAL STUDIED	NO. OF CASES	FINAL ETIOLOGIC DIAGNOSIS							
		CONVENTIONAL CAUSES		ADULT TOXOPLASMOSIS		UNCERTAIN CAUSATION (CONVENTIONAL CAUSES OR TOXOPLASMOSIS)		UNDETERMINED	
		NO.	PER CENT	NO.	PER CENT	NO.	PER CENT	NO.	PER CENT
Preserved serums from previously studied patients	191	80	42	42	22	35	18	34	18
Serums from patients under active study	107	29	27	37	35	12	11	29	27
Totals	298	109	37	79	26	47	16	63	21

crease in incidence of those cases of undetermined causation from 18 to 27 per cent.

The cases attributed either to toxoplasmosis or to uncertain causation (conventional causes or toxoplasmosis) comprise 46 per cent of the patients with granulomatous uveitis. How great is the possibility of error in assuming a toxoplasmic etiologic basis in these two groups? The question is difficult to answer, but since there was a 14 to 30 per cent incidence of positive results in the control group of patients with nongranulomatous uveitis an equally large percentage of unrelated positive results might be expected in the patients with granulomatous uveitis. The actual identity of such unrelated positive test results in granulomatous uveitis cannot be determined from the present information.

Value of Complement Fixation Reaction

Complement fixation reactions were obtained in 92 of the 193 patients. In 30 patients with nongranulomatous uveitis, the result of the complement fixation test was negative in all, although in three

of these patients both the skin and dye tests showed positive results. In 62 patients with granulomatous uveitis in whom the complement fixation test was done, the results were positive in only 7 instances. In each of the 7 cases showing positive reactions, results of both the skin and the dye tests were also positive. However, the complement fixation reaction was positive in only 18 per cent of the 38 patients with both skin and dye test results positive in whom the test was done. Five of these 7 patients had an active exudative chorioretinitis which was ascribed to adult toxoplasmosis; the sixth patient had a chronic granulomatous uveitis believed to be due to toxoplasmosis, while in the seventh patient with a positive complement fixation reaction, the diagnosis lay between tuberculous and toxoplasmic uveitis. The relationship between these 92 complement fixation reactions and the results of the skin and dye tests is shown in table IV.

From these figures it is evident that the complement fixation reaction is of little diagnostic aid in ophthalmic cases. The reaction is quite irregular in its appearance, and apparently it does not remain positive for any prolonged period. A positive complement fixation reaction developing and increasing in titer during the progress of an acute infection is, of course, strong evidence for a toxoplasmic causation. However, in this and in the previous series of patients, such a developing and increasing complement fixation reaction was observed in only two instances. Certainly a negative test result has no significance.

COMMENT

There are a number of interesting questions pertinent to these diagnostic tests which can be at least partially answered by the information available from this study. One of the first questions relates to the specificity of the skin and dye tests. A study of the cutaneous sensitivity to toxoplasmin showed no evidence of any relation between this and positive reactions to tuberculin, brucellin, brucellergin, or streptococcal or other bacterial antigens by which the patients were tested. Likewise, there was no relation between sensitivity to toxoplasmin and to any of the fungus antigens. As concerns both the skin and dye tests, no relation was found between positive reactions to these and any findings in the medical survey, such as skull calcification, eosinophilia, serum globulin, and foci of infection.

Further questions relate to the diagnostic criteria employed. Are they so strict that true cases of ocular toxo-

TABLE IV

RELATION OF TOXOPLASMIN SKIN REACTION AND DYE TEST RESULTS TO COMPLEMENT FIXATION REACTION

RESULTS OF TESTS*	NO. OF CASES	COMPLEMENT FIXATION			
		POSITIVE		NEGATIVE	
		NO.	PER CENT	NO.	PER CENT
Positive S + Positive D	38	7	18	31	82
Negative S + Negative D	23	0	—	23	100
Positive S + Negative D	15	0	—	15	100
Negative S + Positive D	16	0	—	16	100

*S = Skin Test; D = Dye Test

plasmosis are excluded from proper classification, or are they so low that old and unrelated systemic Toxoplasma infections are incorrectly supposed to be the etiologic factor in uveitis?

In answer to this question, it might be pointed out that in the case reported by Jacobs and his co-workers,[9] in which the dye test was done at roughly the same time that the Toxoplasma parasite was demonstrated in the ocular lesion, the titer was 1:64. In the case reported by Duke-Elder and his colleagues,[3] in which the dye test was done twenty-one months after the demonstration of *Toxoplasma* in the eye, the titer was 1:160. In the series of 21 patients reported by Jacobs, Cook, and Wilder,[10] in whose enucleated eyes Wilder had previously found the bodies typical or suggestive of *Toxoplasma*, there were results of dye tests as low as 1:32 two months after the enucleation of the eye. In 2 patients who underwent dye tests two years and eight years after the enucleations, results of the tests were positive in titers of only 1:4 and 1:2 respectively; while in the other patients, for whom the dye tests were done from one to seven years after the enucleation, the titers varied from 1:16 to 1:2048. From these various figures, it is evident that the minimum standard for a significant reaction to a dye test for an adult cannot be greater than 1:64. However, in view of the results reported by Jacobs, Cook, and Wilder, should it be lower? It must be remembered that their figures were from data on patients in whom the dye tests were done some months after the active eye disease. The immediate question is, "What shall be the standard for patients with active ocular disease?" This can be partially answered by reanalyzing the figures of the dye test titers in the patients here reported. If the requirement for a significant reaction to the dye test is lowered to a titer of 1:32 while other criteria are maintained, three additional cases are added to the group of cases with toxoplasmic causation. If lowered to 1:16, two other cases will be added. Thus, the incidence of uveitis attributed to toxoplasmosis would rise from 35 per cent to 38 per cent in the first instance, and to 40 per cent in the latter.

If the criteria are further lowered by omitting the requirement for a positive reaction to the toxoplasmin skin test and at the same time lowering the requirement for a positive result on the dye test, the incidence for toxoplasmosis as the cause of granulomatous uveitis rises to 45 per cent and over with a proportionate drop in the size of groups in which the cause is undetermined. If the requirement for a compatible clinical ocular picture is removed and evidence in favor of other recognized conventional causes of the disease is disregarded, the incidence of endogenous ocular disease which would thus be ascribed to toxoplasmosis on the basis of a 1:64 dye test alone would reach the high figure of 62 per cent; it will soar to still higher levels if the 1:64 titer requirement is lowered. Such interpretations of the frequency of toxoplasmosis as the cause of adult uveitis must be at present regarded as overly enthusiastic. In view of the evidence that proved active ocular toxoplasmosis can occur with dye test titers as low as 1:64, it does not appear possible to raise the standard for a significant test result above this figure. The effect of lowering the standard for a positive test result while maintaining other standards is insignificant. Therefore, until the situation is further clarified, if a diagnosis of adult ocular toxoplasmosis is to be made in any given case of active ocular disease, it would appear unwise to reduce the requirements for a significant result from the dye test to below the present levels, or at least no lower than 1:32.

What is the relation or lack of relation of positive and negative results from dye tests? The incidence of correlation between the results of skin and of dye tests in both nongranulomatous and granulomatous uveitis is shown in table V. Thus, in nongranulomatous uveitis there is correlation, i.e., both test results positive or both test results negative, in 73 per cent of the cases, and in granulomatous uveitis similar correlation in 85 per cent of the cases—statistically comparable figures, with an overall correlation of 80 per cent.

In 20 per cent of the cases, there was no correlation; either the result of the skin test was positive and that of the dye test negative, or *vice versa*. There were 23 such cases in the nongranulomatous group, and 16 such cases in the granulomatous group (table VI). What is the significance of data showing one positive reaction and one negative reaction? It is known that skin sensitivity develops later than serum antibodies, and this explanation might account for some of the discrepant results in individual patients if there is some evidence that the infection had been recently acquired. Not enough proved cases have been followed for a long enough period to allow general statements concerning the relative persistence of serum antibodies and skin reactivity. An individual positive reaction may, conceivably, be an indication either of a toxoplasmic infection or of a cross reaction with some other infectious agent; or it may be due to some technical error. In any event, when the histories of the patients with such unrelated tests are examined, the positive test result appears to be of little significance. In the 23 patients with nongranulomatous uveitis who showed no correlation between skin and dye tests, there was no suggestion of a toxoplasmic causation in any of them. The 16 cases of granulomatous uveitis with uncorrelated test results were distributed as follows: (1) eight cases ascribed to

TABLE V

CASES OF UVEITIS WITH CORRELATION BETWEEN RESULTS OF SKIN AND DYE TESTS

TYPE OF UVEITIS	NO. OF CASES	POSITIVE SKIN AND POSITIVE DYE	NEGATIVE SKIN AND NEGATIVE DYE	TOTAL CORRELATION	
				NO.	PER CENT
Nongranulomatous	86	10	53	63	73
Granulomatous	107	58	33	91	85
Totals	193	68	86	154	80

TABLE VI

CASES OF UVEITIS WITH NO CORRELATION BETWEEN RESULTS OF SKIN AND DYE TESTS

TYPE OF UVEITIS	NO. OF CASES	POSITIVE SKIN AND NEGATIVE DYE	NEGATIVE SKIN AND POSITIVE DYE	NON-CORRELATION	
				NO.	PER CENT
Nongranulomatous	86	16	7	23	27
Granulomatous	107	7	9	16	15
Totals	193	23	16	39	20

conventional causes; (2) seven cases ascribed to the category of undetermined causes; (3) one case of granulomatous uveitis with a superimposed allergic reaction, ascribed to undetermined causation.

This last case was that of a 26-year-old man with a high degree of cutaneous hypersensitivity to toxoplasmin and with the dye test showing positive results only with the undiluted serum. There were no other findings to which the ocular inflammation could be attributed. The ocular picture was that of old discrete glial scars in the fundus with a superimposed allergic reaction, marked retinal and subretinal edema, but no actual exudate. This picture could well have been due to a toxoplasmic pseudocyst rupturing in sensitized tissue — an explanation first offered by Frenkel (1954). Despite the suspicion that the ocular disease was related to toxoplasmosis, because of the lack of correlation, the case was recorded as of undetermined cause.

The fact that there was an over-all correlation between the findings on skin and dye tests (both tests positive or both negative) in 80 per cent of all the cases raises an interesting question. If the reaction to the skin test is positive, what is the expectancy of a positive result on the dye test? This question is of practical importance and is especially pertinent because the skin test is easy to do, the antigen is available from a commercial source,* and no special equipment is necessary. The skin test is therefore well within the range of diagnostic procedures readily done by the practicing ophthalmologist. On the other hand, the dye test is technically difficult, entails the necessity of keeping infected animals in the laboratory, and is available only to comparatively few clinical ophthalmologists.

*It is understood that Eli Lilly and Company, Indianapolis, makes a limited amount of this antigen and distributes it as a public service.

A further analysis of the material on which this report is based gives a reasonable answer to the above question. In this series as a whole (counting both nongranulomatous and granulomatous uveitis), there were 92 patients with positive reactions to skin tests (table II). When the results of the dye tests found in these 92 patients are analyzed according to (A) the Wilmer Ophthalmological Institute (W.O.I.) standard for significance in the dye test (1:64); (B) a minimum titer of 1:32; and (C) a minimum titer of 1:16, the result is as follows:

	PERCENTAGE
A. Incidence of positive results of dye tests (1:64 standard) in positive skin reactors	72
B. Incidence of positive results of dye tests (1:32 titer standard) in positive skin reactors	76
C. Incidence of positive results of dye tests (1:16 titer) in positive skin reactors	90

If only patients with granulomatous uveitis are considered, the figures are even more impressive. There were 66 positive skin reactions in the group with granulomatous uveitis. The incidence of positive dye test reactions by the above A, B, and C standards in these 66 patients was as follows:

	PERCENTAGE
A. Incidence of positive results on dye tests (1:64 standard) in positive skin reactors	85
B. Incidence of positive results on dye tests (1:32 standard) in positive skin reactors	90
C. Incidence of positive results on dye tests (1:16 standard) in positive skin reactors	95

These figures indicate that the toxoplasmin skin test is an excellent screen-

ing test. In granulomatous uveitis, the margin of error varies from 5 to 15 per cent according to the standard used to determine a positive reaction to the dye test.

If these figures are examined for the converse, i.e., the incidence of positive skin test reactions in patients with positive results on dye tests, the results are somewhat different. Thus, as the dye test standard of significance is changed, A to B to C, in the over-all picture (both granulomatous and nongranulomatous uveitis) there will be an increasing number of positive results while the number of positive reactors to skin tests stays constant, thus giving a declining incidence of correlation.

	PERCENTAGE
A. Dye test result positive by 1:64 standard—skin test reaction positive	81
B. Dye test result positive by 1:32 minimum titer—skin test reaction positive	80
C. Dye test result positive by 1:16 minimum titer—skin test reaction positive	76

If only patients with granulomatous uveitis are considered, the incidence of positive skin reactions in patients showing a positive result on dye tests is as follows:

	PERCENTAGE
A. Dye test result positive by 1:64 standard—skin reaction positive	85
B. Dye test result positive by 1:32 minimum standard—skin reaction positive	84
C. Dye test result positive by 1:16 minimum standard—skin reaction positive	78

These figures are obviously only of academic interest. It is interesting, however, that Frenkel in his 1954 report examined his results from skin and dye tests in this manner and found an 87 per cent incidence of positive skin reactions in his patients who showed positive results on the dye test, a figure identical with the findings reported here when the higher standard for significantly positive results on dye tests was used.

Another interesting question concerns the intensity of the reactions to skin tests and the dye tests. Does the intensity of these reactions follow any definite pattern in the course of toxoplasmic uveitis, and has it any clinical significance? An attempt has been made to divide the cases of supposed toxoplasmic uveitis into acute and chronic types and to plot the intensity of the skin and dye tests in these two groups (tables VII and VIII). Such division into acute and chronic cases is difficult and often arbitrary and unreliable. For example, if a patient has old glial scars in the fundus and a superimposed allergic reaction around them, shall his disease be classified as acute or chronic? Moreover, if the cases are divided into too many groups, the number in each group is so small it is meaningless; nevertheless, for what it may be worth, such an analysis has been made.

Table VII shows the distribution of the skin reactions of different intensities in these two groups and, for comparison, in the group of various forms of uveitis not believed due to toxoplasmosis. In the supposed acute form of the disease, three-plus or four-plus skin reactions were present in 10 of 24 cases, an incidence of 41 per cent. In 13 cases of the chronic form of ocular toxoplasmosis and in 12 cases of uncertain origin (chronic toxoplasmosis or conventional causation) there were three instances of this high sensitivity, an incidence of 12 per cent. In the 16 cases of granulomatous uveitis believed to be due to causes other than toxoplasmosis, there were no instances of high sensitivity. How-

TABLE VII

INTENSITY OF SKIN REACTIONS IN VARIOUS TYPES OF UVEITIS

TYPE OF UVEITIS	NO. OF CASES	+	++	+++	++++
Granulomatous of conventional causation	1	0	1	0	0
Grandulomatous of uncertain causation	12	5	7	0	0
Granulomatous of undetermined causation	15	6	9	0	0
Granulomatous attributed to presumed acute toxoplasmosis	24	1	13	7	3
Granulomatous attributed to presumed chronic toxoplasmosis	13	4	6	3	0
Nongranulomatous	26	8	11	5	2

TABLE VIII

INTENSITY OF POSITIVE RESULTS ON DYE TESTS IN VARIOUS FORMS OF UVEITIS

TYPE OF UVEITIS	NO. OF CASES	LESS THAN 1:256	1:256	1:512
Granulomatous of conventional causation	8	4	3	1
Granulomatous of uncertain causation	12	7	2	3
Granulomatous of undetermined causation	10	9	1	0
Granulomatous attributed to presumed acute toxoplasmosis	24	7	4	13
Granulomatous attributed to presumed chronic toxoplasmosis	13	6	4	3
Nongranulomatous	17	11	2	4

ever, in 26 control patients with nongranulomatous uveitis, there were seven instances of high sensitivity, an incidence of 27 per cent. This high degree of reactivity might well be expected in any group of allergic individuals.

Similarly, dye tests of 1:256 or over were present in 17 of 24 cases of acute ocular toxoplasmosis, an incidence of 70 per cent (table VIII). In 25 cases of granulomatous uveitis, either believed due to chronic toxoplasmosis or of uncertain origin, there were 12 cases showing high dye-test titers, an incidence of 48 per cent, while in the 18 cases believed due to causes other than toxoplasmosis, there were 5 high-titer dye tests, an incidence of 21 per cent. In the nongranulomatous control group, there were 6 cases of high titers, an incidence

of 35 per cent. These figures are of little statistical significance and are open to criticism on the basis of the classification of patients. However, they show an apparent higher incidence of strong reactions on skin tests and high-titer results on dye tests in the group of patients supposed to have acute ocular toxoplasmosis. Thus they tend to confirm Frenkel's contention that there are two general patterns of ocular toxoplasmosis, each with a somewhat different type of immune response: (A) Acute infections, in which the reaction to skin test may at first be absent or of low intensity but soon becomes strongly positive. Response to the dye test is usually positive in high titers; results of the complement fixation test are variable, but they may become positive and reach high titers. (B) Chronic infections, when the toxoplasmin test result is positive but only of moderate intensity. Results of the dye test are positive, often in low titers, and those of the complement fixation test are usually negative.

The types of ocular lesions which are believed to be due to ocular toxoplasmosis are shown in table IX. The incidence of these lesions in the patients previously reported (Series I) is also shown in this table. There is little difference in the two series. In the over-all picture, there are 79 cases believed to represent examples of adult ocular toxoplasmosis. Fifty-one of these, or 65 per cent, were cases of the so-called "focal" choroiditis or chorioretinitis. Seven, or 9 per cent, were cases of old granulomatous choroiditis with a superimposed allergic reaction. Seventeen were cases of generalized uveitis, an incidence of 21 per cent. There were four cases of retinal periphlebitis in the first series and none in the second series, an over-all incidence of 5 per cent. However, since July 1, 1955, several cases of retinal periphlebitis believed due to toxoplasmosis have been observed, and there is no reason to modify the opinion that ocular toxoplasmosis may occasionally manifest itself under this clinical picture.

TABLE IX

OCULAR LESIONS OBSERVED IN CASES ATTRIBUTED TO TOXOPLASMOSIS

TYPE OF LESION	NO. OF CASES		OVER-ALL TOTAL
	SERIES I	SERIES II	
Focal chorioretinitis	28	23	51
Disseminated or focal chorioretinitis with superimposed allergic reaction	3	4	7
Generalized uveitis	7	10	17
Periphlebitis retinae	4	0	4
Totals	42	37	79

The last question concerns the effect of treatment on ocular toxoplasmosis. On this point there is but little information to be gleaned from patients here reported. The majority of the cases diagnosed as ocular toxoplasmosis were referred cases, and while treatment was instituted or recommended in every case,

the majority of the patients returned to the referring physicians. Accurate follow-up information has not been obtained in many instances. In some the period of observation after treatment is still too short to permit any conclusions. In all, there are 22 patients with various forms of uveal disease believed to be due to a toxoplasmic infection who have been treated with pyrimethamine (daraprim) and sulfadiazine, and reasonably accurate information is available regarding them (table X).

TABLE X

RESULTS OF TREATMENT OF OCULAR TOXOPLASMOSIS WITH DARAPRIM AND SULFADIAZINE IN 22 CASES

RESULT	NO. OF CASES	EXCELLENT	MODERATE	FAIR	SYSTEMIC REACTIONS
Definite improvement	13	4	4	5	Four minor—leukopenia (2) crystalluria (1) weight loss (1)
Questionable improvement	3	0	0	0	Four severe—agranulocytosis (2) dermatitis and pyrexia (2)
No improvement	6	0	0	0	

Thirteen of these 22 patients showed a favorable response to this therapy in the sense that the inflammatory reaction was speedily controlled. In 5 of these there was little return of vision, due to macular involvement. In 4 there was moderate improvement, and in 4 the improvement was dramatic. In 3 other cases there was questionable improvement in the clinical picture, and in six the treatment was without effect. Frenkel and Friedlander[6a] have shown that the *Toxoplasma gondii* occurs in both proliferative and cyst forms, each with a somewhat different structure. Eyles[2] reported that although the proliferative form of the parasite is vulnerable to therapeutic attack, the encysted form is protected. It is possible that this may explain the variations in the therapeutic response. In infections by the proliferative form, chemotherapy would be effective, but in the acute superimposed allergic reaction believed to be due to the rupture of a pseudocyst in sensitized tissue, chemotherapy would be ineffective and desensitization would theoretically be the proper procedure to reduce the hypersensitive uvea to a nonreactive state. As sufficient toxoplasmin has become available, several patients in this last category have been placed on such a desensitization regimen with the Toxoplasma antigen. It will, however, be some time before the efficacy of this therapeutic attack can be evaluated.

In the administration of daraprim and sulfadiazine, the therapeutic regimen thus far followed has been the administration of 25 mg. of daraprim daily for the first three weeks. Thereafter the drug is administered in the same dosage every second day. Sulfadiazine is given in a total daily dosage of 2 gm. during the period of therapy. Therapy is continued for a minimum of six weeks, if possible, then continued or discontinued on the basis of the observed therapeutic response.

As indicated in table X, there was a large percentage of untoward reactions under this therapy. Four patients in this

series had severe reactions. One of these required several transfusions to recover from agranulocytosis. Two other patients had a severe dermatitis with pyrexia. Four other reactions were relatively insignificant.

SUMMARY AND CONCLUSIONS

1. An analysis of the results of the skin and dye tests for toxoplasmosis in 193 adult patients with uveitis has been made. Eighty-six of these patients had the nongranulomatous form of the disease, and 107 had the granulomatous form. The criteria employed for the diagnosis of ocular toxoplasmosis in the individual case were as follows:

   a. An ocular picture believed compatible with Toxoplasma infection.

   b. The exclusion of other systemic granulomatous infections which could serve as etiologic factors.

   c. The presence of a positive toxoplasmin skin reaction together with positive results of a dye test in a minimum titer of 1:64.

2. Under these criteria, 37 cases, or 35 per cent of the 107 cases of granulomatous choroiditis, were attributed to toxoplasmosis.

3. An analysis of the results of the complement fixation test indicated that this test was of little or no value unless the positive result developed during the course of an acute infection.

4. Although the incidence of a positive diagnosis of ocular toxoplasmosis is not greatly affected by lowering the standards for a significant result from the dye test to positive in titers of 1:32 or even to 1:16, the evidence available from this study indicates that a dye test titer of at least 1:64 may reasonably be expected in an individual with active ocular toxoplasmosis, and that it is at present unwise to reduce the criteria for a significant result below this level.

5. In the over-all picture, with the result of the dye test considered as positive only at titers of 1:64 or higher, there was 80 per cent correlation between the skin test and the dye test, i.e., either the results of both tests were positive or the results of both were negative.

6. There was no correlation, on the 1:64 dye test titer basis, between results of skin tests and dye tests in 16 patients with granulomatous uveitis. If titers of 1:16 were considered positive, there were only 2 patients with granulomatous uveitis who had positive skin reactions and in whom the results of the dye test were negative.

7. In the entire series, there was a 72 per cent incidence of positive results on the dye test among the patients who showed positive skin reactions. When the standards for a positive dye test were lowered to 1:32 and to 1:16, this incidence rose respectively to 76 and to 90 per cent.

8. When granulomatous cases only were considered, this incidence of positive results of the dye test under the three standards (1:64, 1:32, and 1:16) in positive skin reactors was respectively 85 per cent, 90 per cent, and 95 per cent.

9. High-intensity skin reactions and high-titers on the dye tests appeared to be more frequent in the acute forms of ocular toxoplasmosis, although the evidence on these points is inconclusive.

10. In this and in the former series there were a total of 79 cases of ocular disease attributed to toxoplasmosis. These were divided as follows: 51 cases of focal choroiditis or chorioretinitis, 7 cases of old granulomatous chorioretinitis with a secondary acute allergic re-

action superimposed, 17 cases of generalized uveitis, 4 cases of retinal periphlebitis.

11. The effect of treatment with daraprim and sulfadiazine in 22 patients was analyzed. In 13 of these there was a definite therapeutic response. Severe side reactions occurred in 4 patients.

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DISCUSSION

MICHAEL J. HOGAN, M.D., San Francisco: This careful analysis of 200 cases of endogenous uveitis for serologic and skin-test evidence of a toxoplasmic origin helps greatly to bring these tests into their proper perspective. The authors have attempted to evaluate their cases in order to compare the results of the studies on toxoplasmosis with those obtained as a result of a search for other possible causative diseases.

All of you who have managed patients with uveitis, or who have been concerned with investigative work in this field, understand the frequency with which the clinician is unable to arrive at a specific diagnosis in cases of endogenous uveitis. Most often this inability is not due to a lack of cooperation between the physician and patient, but rather it seems to be the result of inadequate clinical and immunologic techniques. Progress in the detection of the etiologic factor in various forms of uveitis has been remarkably slow, and it is little wonder that investigators most often turn to other more rewarding and interesting fields.

It is widely agreed that each new discovery always creates more problems than it solves, and such is the case with toxoplasmosis. As soon as we learned that the disease existed, and that ocular involvement and central nervous system involvement were prominent manifestations, we commenced to speculate that some of the cases of juvenile and adult endogenous uveitis might be due to Toxoplasma. Not an iota of evidence in favor of this hypothesis appeared until the report of Mrs. Wilder, who found organisms resembling Toxoplasma in microscopic sections from over fifty eyes enucleated because of old

uveitis and for other reasons. Many observers agree that these organisms are probably Toxoplasma, and I am inclined to agree with one who said, "What else could they be?" Other very competent investigators state that one cannot draw conclusions with regard to toxoplasmosis from this finding.

The work of Jacobs and his co-workers and that of Habegger seem to me to prove conclusively that Toxoplasma can be implicated as an organism which causes endogenous uveitis. This raises a number of important questions with regard to acquired juvenile and adult ocular toxoplasmosis:

1. *What is the nature of the toxoplasmic ocular lesion or lesions?*

A perusal of the previously mentioned reports suggest that the disease is mainly unilateral; that it commences in the retina and secondarily affects the choroid and, sometimes, the sclera; that it must affect the anterior eye only rarely; that multiple foci of fairly large size occur; that the lesions have a strong tendency to recur over a period of years; and that they produce necrosis in the retina and a granulomatous reaction in the choroid.

2. *What is the possible frequency of such infections?*

One cannot make a positive diagnosis of any infectious disease without the isolation and identification of the causative organism. The rarity with which this has been done in the eye and the limitations of clinical investigative procedures render such a possibility remote. We must, then, fall back on the evidence provided by immunologic procedures to enable us to make even a presumptive diagnosis. The evidence has, up to the present time, been extremely disappointing, and there is even the suggestion, discussed by the authors, that the testing procedures are not entirely valid. A good deal of additional investigation on this aspect of the problem is warranted.

The only conclusion which can be drawn from this and other papers by the authors, and by your discussant, is that the incidence of positive dye tests in patients with chorioretinitis is more than double that in patients with iridocyclitis and in control groups taken from the normal population. This seems to me to be important information, but one cannot draw conclusions from it with regard to the frequency of toxoplasmic uveitis, or with regard to the diagnosis in an individual case.

The skin test also seems to be of little assistance in assessing the incidence of a possible toxoplasmic ocular infection, even though the incidence of positive reactions in granulomatous chorioretinitis is approximately double that in the group with nongranulomatous inflammations.

3. *Can the dye and skin tests be used to advantage in assisting the diagnosis?*

a. The dye test. Numerous articles have been written attempting to prove that titers of various levels have significance. I think it is best to consider that any titer over 1:32 may have some significance, provided that no other etiologic factor can be determined, and the ocular manifestations are consistent. I do not think one should attempt to make the diagnosis of toxoplasmic chorioretinitis on the basis of any particular titer level, but one should keep in mind the possibility that a positive titer may have some significance. A titer which increases or decreases significantly over a period of months may provide diagnostic information.

The authors found 53 patients with positive dye test reactions who had negative results on skin tests. This finding is not dissimilar to that encountered in our own series of patients, and it emphasizes the importance of not overestimating the value of these tests.

b. The skin test. This test has been found to be positive in 30 to 40 per cent of normal adults in the population of the United States. In the authors' report, the results for the group with granulomatous chorioretinitis indicate that approximately 60 per cent had positive reactions. The variations in antigens used, methods of skin testing, controls, and the criteria accepted for positive reactions all influence the results obtained by various authors, and such variations may account for some of the 13 per cent of patients reported here with positive skin reactions and negative results on dye tests. It is to be hoped that antigens and controls for skin testing will be standardized to provide more uniform results.

The diagnosis of granulomatous versus nongranulomatous uveitis, especially of the posterior type, has always proved extremely difficult for us, and enquiry among other ophthalmologists reveals a similar difficulty. Therefore I feel that it is better to consider these results in the light of a simple posterior or anterior uveitis. According to my figures, 80 per cent of the reported cases in the authors' series had a posterior or diffuse uveitis; and of these, 81 or 55 per cent showed positive results on the methylene blue dye tests. For the same reason, I feel that the

results in the toxoplasmin and streptococcal skin tests may be altered by this system of classification.

With regard to therapy with daraprim and sulfadiazine, the failure of a possible ocular lesion to respond to this type of therapy might be due to the fact that the organisms are obligate intracellular parasites and might not be affected by these chemicals. Also, in the authors' cases the dosage of drugs might have been insufficient. I realize the dangers which might ensue from larger doses used over such a long period of time. We have treated 13 cases of possible toxoplasmic chorioretinitis with 75 mg. of daraprim and 4 grams of sulfadiazine daily for 14 to 21 days, and we obtained very equivocal results. One case with a very high titer and severe bilateral chorioretinitis failed to respond to this method of therapy.

It has been suggested by some observers that failure to respond to therapy with sulfadiazine and daraprim might be used as a therapeutic test to rule out a possible ocular toxoplasmosis. I would advise against such a method of trying to establish a diagnosis. There is great danger involved in the use of these drugs, especially in children.

I would like to ask the authors whether they had a chance to investigate the seven eyes which were enucleated for a possible etiologic factor.

REPAIR OF PTOSIS USING FRONTALIS MUSCLE AND FASCIA LATA

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ALTHOUGH it is generally conceded that no single type of ptosis operation will serve for the best correction of all cases, it has been the experience of the surgeons at the Hospital for Sick Children, Toronto, that in most of the ptosis cases seen during the last four years there was very little or no levator action and they were best suited to repair by using the frontalis muscle and fascia lata.

The ideal procedure would appear to be one of the many operations designed to shorten, and hence utilize, the action of the levator muscle. Good results are obtained with this method by surgeons who repair a large number of ptosis cases, but most oculists, like myself, do not treat enough of these patients to obtain uniformly good results. Most of us would prefer to adopt the simplest method giving the best correction. It is with this thought that most of our ptosis cases have been repaired with fascia lata. Many surgeons avoid the operation because of their inexperience in taking fascia and of the problems involved in having a general surgeon take it for them.

It is the purpose of this paper to describe a technique for taking fascia and a technique for its use in suspending the drooped lid. The method of ptosis repair is essentially that described by W. W. Wright in 1922,[2] with several minor modifications. Derby described a similar procedure in 1928.[1]

During the period July 1, 1951, to June 30, 1955, 102 patients at the Hospital for Sick Children had ptosis operations. Eighty-five of these children had the corrective operation done with fascia lata (33 of the operations were bilateral), 16 had their lids elevated by levator resection (four of the corrections were bilateral), and one had a tarsal resection. In 89 cases the ptosis was congenital; in five it was post-traumatic; in four it was the result of progressive nuclear ophthalmoplegia; and in four it was due to neurofibromatosis.

Forty-nine of the 102 patients with ptosis had little or no levator action, 36 had only fair levator action, and 17 had fairly good levator action. Of this latter group of 17 patients, 16 were chosen to receive levator resection and one to receive tarsal resection. Table I shows the ages at which the various operations were done.

TECHNIQUE OF OPERATION

Taking the Fascia

The leg is prepared with green soap, iodine and alcohol. Shaving the thigh is not necessary in children. The skin incision is made on a line between the lateral condyle of the tibia and the anterior superior iliac spine, starting approximately two inches above the knee and extending upwards one and one-half inches (fig. 1, A). The incision is deepened

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TABLE I

AGES AT WHICH THE VARIOUS TYPES OF OPERATION WERE DONE IN 102 PATIENTS WITH PTOSIS

OPERATION	AGE IN YEARS													TOTAL
	2	3	4	5	6	7	8	9	10	11	12	13	14	
Fascia lata repair	18	19	15	7	5		3	2	1	4	2	3	1	85
Levator resection	5	1	2	5	2	1								16
Tarsal resection				1										1

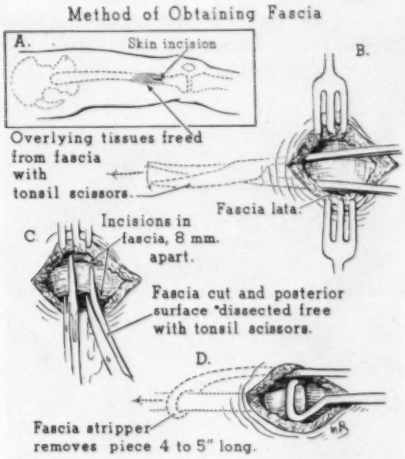

Method of Obtaining Fascia
A.
Skin incision
B.
Overlying tissues freed from fascia with tonsil scissors.
Fascia lata.
C
Incisions in fascia, 8 mm. apart.
Fascia cut and posterior surface dissected free with tonsil scissors.
D.
Fascia stripper removes piece 4 to 5" long.

FIG. 1—Method of obtaining fascia from thigh.

until the fascia comes into view. A pair of long tonsil scissors is passed upwards with a spreading action (not cutting) to free the fascia from the subcutaneous tissue (fig. 1, B). Two vertical incisions are now made in the fascia, 8 mm. apart, with a scalpel. One blade of the tonsil scissors is passed through the incision, and the scissors are pushed upwards with the blades kept approximately 15 degrees apart. The fascia is thus divided along the direction of its fibers. The procedure is repeated in the other fascial incision. The lower end of the fascia is cut across (fig. 1, C), and the scissors are then passed upwards, freeing the fascia from the underlying muscles. The lower end of the fascia is passed through the circular end of the fascia splitter, and the end of the fascia is held with a forceps (fig. 3). The fascia stripper is passed upwards for a distance of four to five inches; then the fascia is cut (fig. 1, D). Dividing the fascia into 3 mm. strips is easiest done on a board after the fascia is spread out evenly. Straight Stevens scissors are used for this purpose. Two fascia strips are used for each lid.

Placing the Fascia in the Upper Lid

Three skin incisions are made in the upper lid 2 mm. from the lid margin. The lid is first marked into thirds. The first incision is made parallel to the lid margin in the center of the lid, extending laterally and medially to within 1.5 mm. of the marks. The second incision is made medial to the medial mark on the lid, starting 1.5 mm. medial to this mark. The incision is made approximately 4 mm. long. The third incision is made lateral to the outer mark on the lid, starting 1.5 mm. lateral to this mark, and the incision is made approximately 4 mm. long (fig. 2, A).

The incisions in the forehead (fig. 2, A) are made 7 mm. above the brow and parallel to it, the outer one being placed 6 mm. lateral and the inner one placed 6 mm. medial to the corresponding incisions near the lid margin. A third inci-

sion is made in the brow 6 mm. above and parallel to the other incisions, lying midway between them. The Wright fascia needle (fig. 3) is passed down

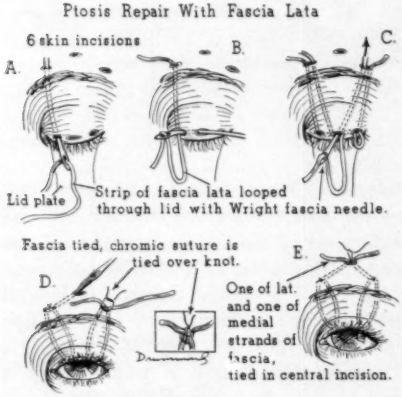

Ptosis Repair With Fascia Lata
6 skin incisions
A.
B.
C.
Lid plate
Strip of fascia lata looped through lid with Wright fascia needle.
Fascia tied, chromic suture is tied over knot.
D.
E.
One of lat. and one of medial strands of fascia, tied in central incision.

FIG. 2.—Ptosis repair with fascia lata.

through the lateral incision in the brow, emerging from the lateral incision in the lid margin (fig. 2, A). A lid plate is placed under the upper lid margin in order to protect the globe. The fascia is threaded through the hole in the needle and drawn upwards. The needle is now passed into the center incision in the lid margin and emerges through the lateral incision.

The lower end of the fascia, after being threaded through the eye of the needle, is drawn under the 3 mm. bridge of skin. The fascia needle is again passed through the lateral brow incision, but this time it is brought out through the center incision in the lid margin. The fascia is passed through the eye of the needle and drawn upwards. The second piece of fascia is placed in the medial side of the lid in a similar fashion (fig. 2, B and 2, C).

Before the upper ends of the fascia are tied, a piece of 3-0 chromic suture is laid beneath them. The first loop in the fascia is tied, then the chromic suture is tied about this to prevent the fascia from slipping. The second loop in the fascia is tied and the chromic suture is again tied around this (fig. 2, D). The lid is pulled up 2 mm. more than desired for the final result. The other piece of fascia is pulled up and tied in a similar manner. One end of the fascia above the knot on the lateral side is pulled up under the skin with the fascia needle and out through the upper center incision. An end of the fascia above the medial knot is also pulled under the skin and out the same incision (fig. 2, E). These ends

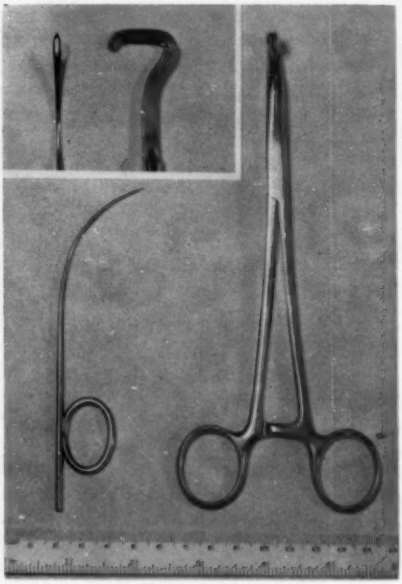
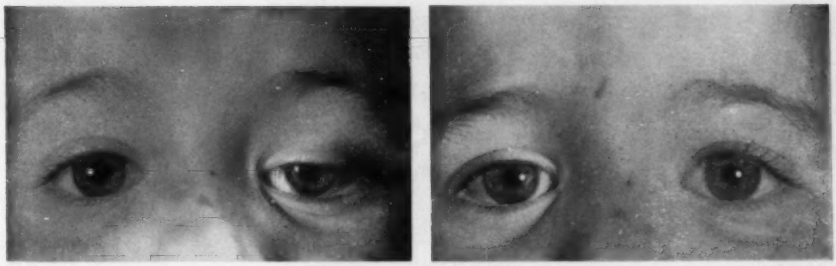


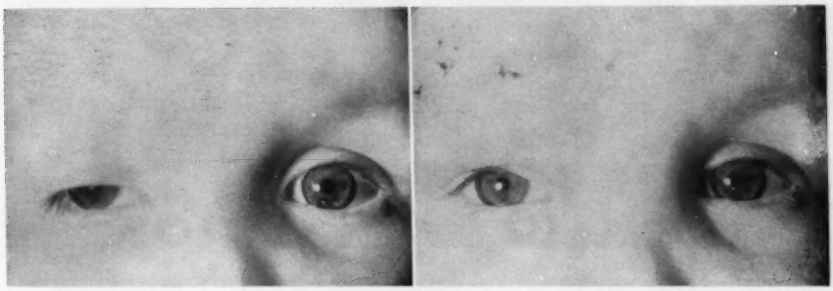
FIG. 3.—Wright fascia needle (left) and forceps for holding fascia (right).

are tied, with chromic catgut securing the knot. This prevents the fascia from slipping down. The skin incisions are closed with an end of the chromic suture or plain catgut so that these sutures do not have to be removed at a later date,



A B

FIG. 4—W. H., age five. (A) Congenital ptosis left eye. (B) Three months after fascia lata repair of ptosis.



A B

FIG. 5—M. C., age three. (A) Congenital ptosis right eye. (B) Two weeks after fascia lata repair of ptosis.

which is sometimes difficult in children. One or two modified Frost sutures of black silk are placed through the lower lid, being brought out through the skin of the brow and tied over a button. These protect the cornea and are removed on the second or third day.

RESULTS OF OPERATION

Of the 85 cases repaired with fascia lata, good results were obtained in 76. Of the remaining 9 patients, 4 were improved sufficiently to satisfy the parents with the results, and no further surgery was done. In the other 5 cases the operation was repeated with fascia lata and good results were obtained. Fifty-two, or 61 per cent, of the cases repaired with the fascia were done between the ages of two and four years (table I). It is difficult to obtain enough fascia before the age of two due to the short length of the thigh.

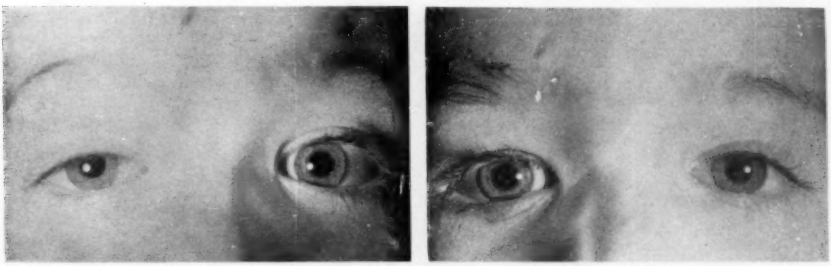
Nine of the 16 patients having levator resection obtained good results. Four of the remainder later had fascia repair, and in the remaining 4 no further surgery was done. Figures 4 to 7 are preoperative and postoperative pictures of patients having fascia lata repair.

CONCLUSIONS

At the Hospital for Sick Children a much higher percentage of satisfactory results was obtained when fascia lata was used in ptosis repair. This applied to all types of ptosis, including those with good levator action. The largest percentage of cases in the series had limited levator action and so were best suited to this type of operation.

A B

FIG. 6—R. R., age six. (A) Congenital ptosis right eye. (B) Five months after fascia lata repair of ptosis.



A B

FIG. 7—D. F., age four. (A) Congenital ptosis right eye. (B) Three weeks after fascia lata repair of ptosis.

The following advantages accrue from ptosis repair with fascia:

1. If the points mentioned in the taking of fascia are followed, the operation is relatively simple.
2. The operation can be done as soon as the thigh is long enough for sufficient fascia to be obtained, i.e., when the child is about two years of age.
3. A good lid fold matching the fellow eye is produced.
4. The operation can be done in cases in which a poor result has been obtained with levator resection.
5. The postoperative swelling is much less than in levator resection, and the child may be discharged sooner from hospital.
6. The advantage of using fascia instead of silk is the absence of delayed infection. Infection may occur immediately after the operation, but it is readily controlled with antibiotics. When silk is used, infection may occur months or years later.

Disadvantage of the operation:

1. Lagophthalmos is present during sleep.

Most parents mention this immediately after the operation, but in a matter of two or three months the lid usually covers the cornea during sleep. The "suspended" appearance of the lid gradually disappears and the patient is able to raise and lower the lid very well as he learns to control the frontalis muscle.

SUMMARY

The technique of placing the fascia in the upper lid, as originally described in 1922 by W. W. Wright, has been described with several minor modifications.

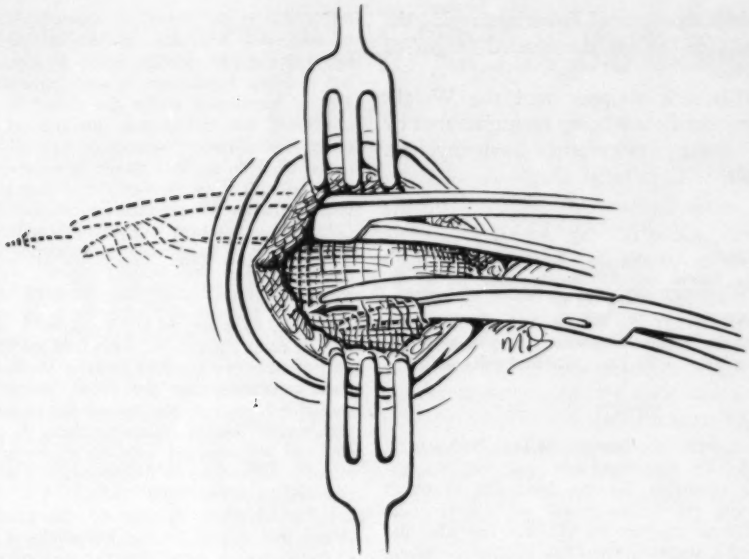


FIG. 8—Method of dividing the fascia fibers, using long tonsil scissors with blades kept approximately 15 degrees apart.

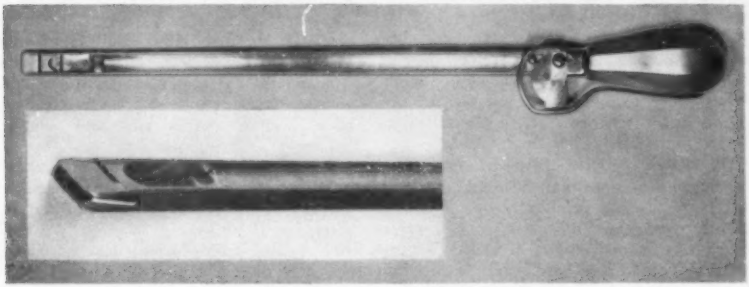


FIG. 9—New fascia stripper.

If the fascia is freed by blunt dissection from the connective tissue above and below and then divided vertically before the fascia stripper is applied, this part of the operation is greatly simplified.

During the four-year period July 1, 1951, to June 30, 1955, a total of 102 cases of ptosis were repaired by various methods, and the results have been reported. Fascia lata repair is the method that appears to be suitable in most cases.

ADDENDUM

I have developed a new fascia stripper which is based on the principle that if a long pair of tonsil scissors is pushed up to divide the fibers of the fascia with the blades kept about 15 degrees apart (fig. 8), it is much easier to pass up the old-type strippers to cut off the fascia. A *V* has been made on each side of the head of the new stripper corresponding to the open scissor blades (fig. 9). A cutting

blade is then moved down by turning the handle to cut off the desired length of fascia.

This new stripper and the Wright fascia needle are being manufactured by the Storz Instrument Company, St. Louis.

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1. Derby, George S.: Correction of ptosis by fascia lata hammock, Am. J. Ophth., 11:352-354 (May) 1928.
2. Wright, W. W.: The use of living sutures in the treatment of ptosis. Arch. Ophth., 51:99-102 (March-April) 1922.

DISCUSSION

RAYNOLD N. BERKE, M.D., Hackensack, N. J.: Dr. Crawford has just described another operation for the treatment of ptosis utilizing the lifting power of the frontalis muscle to support the lid. He has also designed a simple method for getting the fascia lata from the thigh, a method which we as ophthalmologists should not find too difficult. In selecting the fascia lata to produce adhesions between the lid and the brow I think he has shown good surgical judgment, because this material is better tolerated in the tissues of the upper lid than some other materials. For example, if we use skin of the upper lid under the brow to support the lid, we know that cysts often form. Also, if orbicularis muscle of the upper lid is buried under the brow in a similar fashion, it is only a matter of time before those muscle strips stretch out and the immediate postoperative result of the operation is neutralized. Moreover, if we use a foreign material, such as silk, catgut, nylon, tantalum wire, stainless steel, platinum wire, or kangaroo tendon—all of these substances have been used—we know that such substances have a tendency to pull through the tissue or to become infected in time, as Dr. Crawford has just mentioned. For this reason I think that his selection of fascia lata for this purpose is a happy choice.

All the operations which suspend the lid from the brow unfortunately have two disadvantages. For example, if we are successful in correcting the ptosis in the primary position, as Dr. Crawford has so excellently shown us in his cases, then there is always lagophthalmos in looking down and ptosis in looking up unless the patient raises his brow and corrugates his forehead. In binocular ptosis this is not a serious cosmetic blemish, because the lids are in similar positions whether the eyes are up, level, or down, and the cosmetic appearance is quite satisfactory. But in monocular ptosis the defect is quite noticeable. For this reason, this type of operation should not be advocated for all types of ptosis. I do think it should be reserved for cases in which resection of the levator is contraindicated or has resulted in failure.

In this connection it should be added that as we grow in experience with the operations for shortening the levator muscle, we find fewer and fewer cases that result in failure.

W. W. WRIGHT, M.D., Toronto: I should like to congratulate Dr. Crawford on his report of over one hundred cases of ptosis operated on during the past four years—with special reference to the use of fascial sutures and his method of obtaining them. I should also like to commend what he modestly calls minor modifications in the operation originally reported by me in 1922.

I can strongly recommend the operation as one that fulfills the two essentials—a good cosmetic and a good functional result—and also as one that has stood the test of time. I have recently seen two cases that were operated on some thirty years ago and the results were even better than shortly after the operation, as the inevitable slight lagophthalmos tends to decrease with time.

I should like to stress the fact that autogenous fascial sutures, unlike transplanted bone, become vascularized and remain permanently as living tissue.

The use of fascial sutures, now a common practice in general, orthopedic and plastic surgery, was developed in Toronto shortly after the First World War by W. E. Gallie and Arthur LeMesurier, who carried out a series of animal experiments over a period of two years which proved that the fascia became living tissue. These men, still active, are well known among surgeons in this country as well as in Canada.

In conclusion, I should like to issue three warnings. First, do not attempt to improve the operation by putting the sutures into the tarsus, as this tends to draw the lid up toward the brow instead of letting the tarsus slide back into its normal position; second, do not try to save the effort of getting autogenous fascia by using pickled fascia lata, for this defeats one of the main objects of the operation—that of getting living tissue instead of a foreign body; third, fascia lata is very slippery, and knots tied with it will loosen unless the knots are tied over with catgut as described by Dr. Crawford.

SOME BASIC CAUSES FOR FAILURE IN GRAFTING SKIN AND MUCOUS MEMBRANE TO THE LIDS AND SOCKET

MILTON SINGER, M.D.
PORTLAND, OREGON

ANYONE who has performed skin or mucous membrane grafting has had his share of successes, and some failures. However, the failures are rarely recorded in as great detail as are the successes. We are all aware of the many conditions which are necessary for successful results, but it is of value to reconsider some of the basic factors in grafting and why our grafts may fail. Many of the reasons for failure are common both to skin and to mucous membrane grafting, and some are specific only for one or the other.

The general condition of the patient is important. If the patient is chronically debilitated or acutely ill, the chances for a successful take are markedly reduced. When surgery is elective, all underlying chronic or acute ailments should be adequately treated before grafting. If grafting is of an emergency nature, as in burns, one must use all possible supportive measures, such as whole blood, plasma, and morphine, to save the patient's life.[11] When large areas of the body are burned, split-skin grafts may be lifesaving measures alone.

Even a very minor infection may result in failure of a graft to take. When there has been obvious gross contamination, preoperative therapy with wet dressings, antibiotics, or chemotherapy is essential until infection has been eliminated. It has been shown[7] that so-called clean wounds are contaminated with many bacteria, Staphylococcus predominantly. When contamination is suspected, cultures may be taken. A rare infection following wounds about the face and orbit is gas gangrene. It has also been pointed out that tetanus following ocular wounds is very rare.[2,6,19] When a wound is believed to be contaminated with tetanus, the antitoxin is necessary to create passive immunity, because the incubation period for tetanus is less than the time required for active immunization with tetanus toxoid.[18] When active immunity is known to exist, a toxoid booster is adequate.

TYPES AND CHOICE OF SKIN GRAFTS

It is important to select the proper type of graft for the particular situation in each case. In certain conditions a full-thickness graft is more suitable, in others a split-thickness graft. A full-thickness or Le Fort Wolfe graft includes the epidermis, the dermis, and perhaps some of the underlying subcutaneous fat. A split-thickness or Thiersch graft is taken from the epidermic layer only. There is an intermediate type of graft, the three-fourths-thickness type, which includes epidermis and part of the dermis. It has almost all of the advantages of a full-thickness graft, and it ensures complete removal of the underlying fat (fig. 1).

When skin is cut, it is released from the tensions of surrounding tissues, and the elastic fibers in the dermis cause a primary contraction. Because there are more elastic fibers in a thick graft, this type contracts the most. In the skin of young patients, which is richer in elastic

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tissue, one sees greater contraction than in the skin of older patients. Secondary contraction is due to the maturing scar between the graft and its recipient bed. This is a gradual contraction continuing over many weeks.

A split-thickness graft gives a fairly good cosmetic result on the face, but it is not as good as a full-thickness graft. It is thinner, less flexible, and it does not have the same general appearance and texture as a full-thickness graft. The split-thickness variety is easily obtainable, and it is the only type that can be used on granulating surfaces. It will even take over bare bone. It is less apt to be destroyed by minor infection.[9] When the recipient bed is infected, no matter how minor the infection, a full-thickness graft will not take. The Pseudomonas aeruginosa is an organism which is very difficult to eradicate from the skin, and it is a specific handicap in grafting.

Free grafts tend to develop pigmentary changes, and the split-thickness type changes color even more than the full-thickness type. These pigmentary changes are most noticeable when skin is transplanted from distant areas, such as the abdomen or thigh.

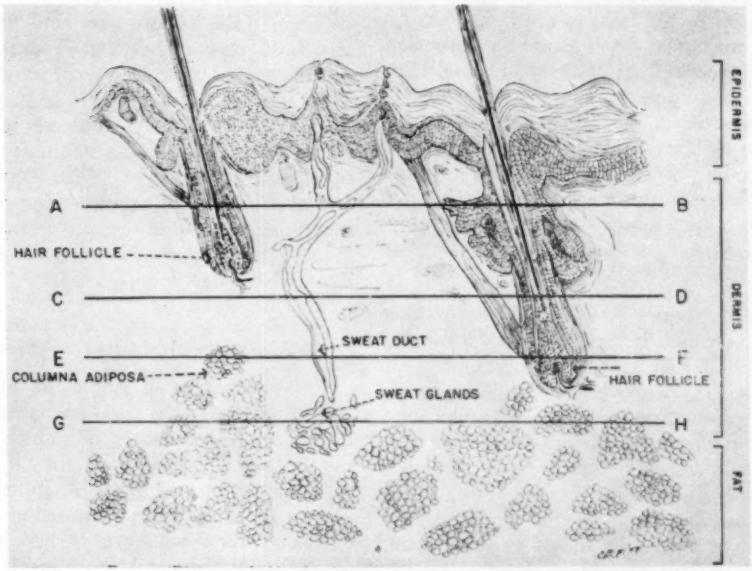

FIG. 1—Diagram of skin showing levels at which skin grafts are removed. AB, level of a Thiersch graft; CD, level of an intermediate split-graft; EF, level of a thick graft; GH, level of a full-thickness skin graft.

In any case in which a free skin graft is applied about the eyelids, and particularly below the lower lid, it is best to create intermarginal adhesions. These adhesions serve a threefold purpose: (1) protection of the cornea, (2) immobilization of the lids, and (3) reduction of graft contraction (fig. 2).

When it is necessary to replace only the skin-muscle layer of the lid, the sur-

geon has an alternative. He may use a local flap created by sliding, rotating, advancing, and transposing, or he may use a free graft. If there has been a large wound, it may be possible to replace all of either lid by double advancement flaps arising from the skin surrounding the same or opposing lid. If the circulation is impaired, flap types of grafts are probably best in order to supply the necessary blood vessels from their bases. When the circulation of the adjoining tarsoconjunctival layer is not disturbed, a free skin graft instead of a circulating flap may be the procedure of choice.[17]

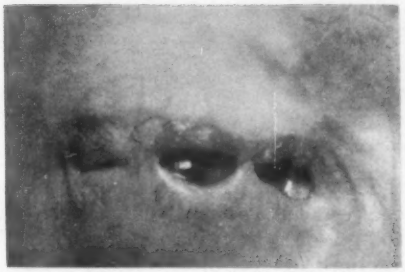

FIG. 2—Split-thickness graft from forearm. Intermarginal adhesions formed after contraction began.

CHOICE OF DONOR SITES

Improper selection of donor skin may jeopardize the cosmetic effect. It is generally felt that a full thickness of lid skin from the same or the opposite side is the most desirable graft. It has better consistency, color, and texture than a graft taken from any other part of the body.[16]

The second best donor site is the cephaloauricular angle. Third, the skin of the supraclavicular triangle is preferred. Most authorities agree on this order of selection, but beyond this there is considerable variation of opinion. A split-skin graft taken from a non-hair-bearing area represents the final choice. Full-thickness grafts from the abdomen, chest, and the extremities are too thick for lid-skin substitutes. Skin from the trunk and extremities may be used as free split-thickness grafts, but the color is usually abnormal.

It is felt that when eyelid skin is taken, it should be cut the same size or just a trifle larger than the recipient bed. Grafts from all other areas should be taken at least one-fourth and preferably one-third larger.[5] If the graft has to be folded or has to fill in a depression, it should be taken somewhat larger than the recipient area. If split skin is to be used to reconstruct a socket, it should be rather large and may be placed around a mold, which can then be applied into the socket.

In badly scarred areas the blood supply may have been impaired so greatly that it will necessitate a pedicle graft rather than a free skin graft.

SURGICAL TECHNIQUE

Improper surgical technique may result in graft failures. Before making the skin incision for a graft, one should study the lines of tension in order to avoid cutting across them. The skin incision should be made at right angles to the skin surface so that the edges may meet perpendicularly when they are sutured. In hair-bearing areas an oblique incision is necessary to avoid cutting across the hair follicles. This is particularly essential when cutting into the eyebrows in order to avoid a hairless scar. Where possible, skin grafts should be handled by sutures passed through the corners of the graft or into the underlying subcutaneous tissues, or by small skin hooks. Forceps, if used, should be fine-toothed in order to avoid crushing the cells on the margin of the skin graft. Forceps without teeth require greater pressure and therefore crush the tissues, with more damage to the cells.

The Nature and Preparation of the Recipient Bed

Inadequate preparation of the recipient bed may result in failure. The first important step is to excise all scar tissue and any other abnormal tissues. Scar tissue is extremely avascular and forms an unsatisfactory foundation for penetration of the graft by new capillaries from the underlying tissues. Such capillary penetration is essential for success. Late secondary contraction is avoided by excision of as much scar tissue in the bed as possible. One may stand helplessly by and watch a skin graft contract beyond repair when there has been inadequate excision of scar tissue (fig. 3). If extensive injury has occurred

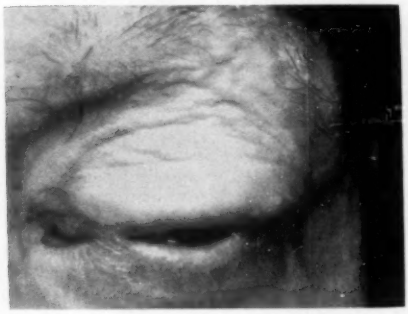

FIG. 3—Contraction of split-thickness graft with wrinkling. Intermarginal adhesions created at the time graft was applied.

and debridement is necessary, it should be extremely conservative. It should consist of the removal of hopelessly devitalized tissue only. It is better to save tissue that may die later than to remove tissue that might live if it were given a chance.

The management of granulation tissue depends upon its nature. If the granulations are flat and nonexudative, it is unnecessary to excise them. If the granulations are hypertrophic, edematous, cyanotic, and exudative, removal of the outer layers is preferable. When the granulations are fibrotic, they should be removed entirely.

Bleeding within the bed may be adequately controlled by simple pressure. If this is inadequate, one may saturate gauze with adrenalin; however, it is best to remember that adrenalin with cyclopropane anesthesia may result in cardiac arrhythmias and even ventricular fibrillation. As a substitute, fibrin soaked in thrombin applied with pressure, or merely the use of a fine-tipped hemostat, may be adequate. At times it may be necessary to touch the fine-tipped clamp applied to a vessel with a coagulating current. Too great coagulation will cause tissue destruction with eventual sloughing. A ligature should be used as a final measure, but it is to be avoided if possible because it introduces a foreign body beneath the free graft.

The graft should fit snugly into the bed without stretching, particularly a full-thickness graft. A split-thickness or intermediate type of graft need not approximate as well. These may overlap the periphery of the bed and be trimmed after the main body of the graft is sutured into place. Interrupted sutures seem to be the most satisfactory, since they adjust only one small portion of the skin edge at a time. One should tie the knot on the side of the host tissue to avoid sloughing of the graft. Close approximation of the tissues with slight eversion of the skin edges avoids formation of dead space. Also, the more accurately skin edges are approximated, the less scar tissue will be formed between them and the finer will be the external scar. The habit of sprinkling antibiotics or sulfa powder into the recipient bed is to be avoided because the powder may cake, producing a foreign body reaction with thick scar formation or keloids.

Pressure Dressing

An adequate pressure dressing is absolutely essential for the success of a skin graft. It is important in order to immobilize the skin graft in its bed and to splint the lids. The pressure should be firm but not strangulating, and too little pressure is of no value. With inadequate pressure the graft may be lifted away from its bed and the new blood

FIG. 4—Cyanosis of graft with loss of superficial epithelium due to too much pressure.

vessels beneath it may be torn, permitting blood or serum to accumulate with ultimate total destruction of the graft, or there may be only localized areas of necrosis due to localized accumulations of blood or fluid. Also, too little pressure may permit fibroblastic proliferation between the graft and its bed, with the formation of a thick scar and ultimate contraction of the graft. Adequate pressure will prevent swelling of the tissues and will control minor hemorrhages as well. Too much pressure may cause impairment of circulation with necrosis of the graft (fig. 4). Over pedicle flaps no pressure is desired. A pedicle may be protected by rolls of gauze on either side of it and a light dressing may be applied over this.

Once a pressure dressing is applied, it should not be disturbed for approximately five to seven days; then the dressing must be removed with extreme care. Adherence of the dressing materials to the skin graft may be avoided by the use of covering materials such as cilkloid or nylon. Ointments over grafts tend to macerate the tissues and inhibit healing. After the first dressing, subsequent dressings may be performed at intervals of two to five days. It is not always necessary to remove the dressing in order to diagnose infection. This is often manifest by temperature elevation, pain, discharge at the edges of the dressing, or foul odor. When an unusual degree of discomfort is complained of by the patient, and when it is deemed absolutely necessary, the pressure dressing may be removed before five days and the wound inspected (fig. 5). Corneal abrasions from inturned or loose lashes are common causes of pain. Sutures, rubber plates, baby buttons, and gauze pledgets, may cause necrosis of the underlying skin within five days, and these should be removed when undue pressure is evident at the first dressing. After four weeks, one may institute gentle emollient massage in an attempt to soften up scars. Vigorous massage too early may cause superficial or deep separation of the tissues.

FIG. 5—Contraction and necrosis of split-thickness graft secondary to purulent infection.

SKIN HOMOGRAFTS

The use of skin from the same species for grafting has been attempted for many years. Generally these grafts have not been successful, and eventually they are destroyed. The skin grafts slough following a process of inflammatory reaction, vascular breakdown, and leukocytic invasion.

In 1953, Romanes[12] reported his experience with the use of homogenous skin in split thickness for repair of symblepharon. The skin was obtained from patients varying in age from 9 to 60 years, and it was refrigerated for six to eight weeks before being used. At the end of two weeks the skin grafts began to slough gradually with concomitant ingrowth of conjunctiva. Romanes and his co-workers made no effort to avoid the sloughing, since the desired effect was replacement of skin by encroaching conjunctiva. Romanes reported 10 cases wherein ultimate replacement by satisfactory conjunctiva occurred. None of the 10 developed any shrinking of the conjunctival fornices.[13] Romanes states that such homografts are of value, since they are easily obtained; they last for a period of two to three weeks, and the eyes remain quiet in their postoperative period. There is a minimum of scar tissue formation, and that which is formed lies on the sclera and episclera where shrinkage is well tolerated. Romanes notes that the use of such skin homografts results in very few complications, and subsequent to his initial report he has continued to use this method for repair of symblepharon with success.[13]

SYMBLEPHARON REPAIR AND CONJUNCTIVAL REPLACEMENT

When there has been conjunctival destruction by burns, by trauma, or by other means, one cannot avoid symblepharon formation merely by the use of a glass rod passed into the fornices daily. Nor can one prevent symblepharon by the use of a conformer alone. It is best to excise all necrotic conjunctival tissue and to replace it with a conjunctival substitute immediately. Merely rearranging conjunctiva that remains may not be enough, and the old practice of a pedicle of skin for replacement is not satisfactory.

Many different tissues have been used to replace conjunctiva, but the best material is conjunctiva itself. Since there is very little conjunctiva available, the next best substitute is buccal mucosa.[14] This must be removed in very thin sheets without the submucosal fat. It is well to mark the upper surface of the conjunctiva or buccal mucosa with a fine suture so that this side is not placed downwards against sclera. All grafts of conjunctiva or mucous membrane must be sutured firmly to the episcleral tissues.

If severe burns have caused complete destruction of the perilimbal circulation and the development of a porcelain appearance, early grafting is essential. This not only restores conjunctiva but improves circulation and nutrition to the cornea. If established symblepharon has developed, all thick scar tissue and granulation tissue must be removed before a graft is applied. If a fornix is being reconstructed, the mucous membrane must be applied well into the fornix and held there with a mold or conformer.

AMNION

Amnion as a replacement was first reported by deRoetth.[14] His materials were obtained by cesarian section from Wassermann-negative persons, and they were stored in tepid Locke's solution before being used. Tissues were sutured to the rectus muscles in order to avoid too much shrinkage, and a contact lens was used over the amnion. Nothing hap-

pened for the first three weeks; however, in the fourth week shrinkage began and continued until the tenth week. The end results were considered poor because of the shrinkage, but the membranes that were left seemed to be transformed to conjunctiva histologically and otherwise. It was felt that the amniotic membrane did not stimulate conjunctival growth but acted as a type of internal splint for newly proliferating conjunctiva. DeRoetth felt that he would use fetal membranes in plastic repair of conjunctiva only when mucous membrane of the mouth was not available.

SKIN FOR REPLACEMENT OF CONJUNCTIVA

Split-skin grafts for conjunctival replacement are used primarily for reconstruction of the socket when the eyeball is absent. When an eyeball is present, irritation of the cornea with ulceration and sloughing may result secondary to the trauma from epidermis or from fine hairs.

If one chooses a skin graft to line a socket, it is best to remove all conjunctiva and to reline the entire socket with skin.[1] It is best to use only the epidermis in split-thickness skin to line a socket, since anything thicker will contain glands and hair follicles which will be accompanied by discharge, growth of hair, or maceration of the skin. As in symblepharon repair, it is necessary to remove all scar tissue and granulation tissue to restore a normal bed before reconstruction can be successful. In this way postoperative contraction of the socket or of the fornices can be avoided (fig. 6).

SUMMARY

An attempt has been made to outline some of the causes for success and failure in grafting skin and mucous membrane. Some attention has been focused on the use of homografts and substitutes for mucous membrane, such as amnion. It is hoped that this review will help, by its reiteration of basic principles, our future success in grafting.

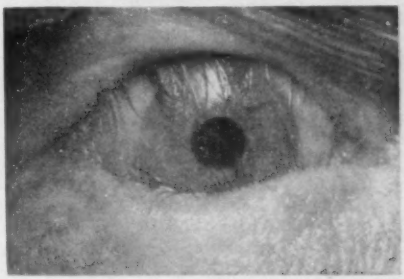

FIG. 6—Inversion of upper and lower lid due to marked contraction of split-thickness skin applied to socket. Inadequate removal of scar tissue prior to graft insertion.

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DISCUSSION

MERRILL J. REEH, M.D., Portland, Oregon: I believe that there is one point that can be re-emphasized: for a source of full-thickness grafts, the skin of the lids is the best. Other sources, such as the back of the ear, are reasonably good substitutes. So often we see grafts which are taken from other portions of the body. Not only are they too heavy and contract too much, frequently they result in an extremely bad match. In dark-skinned people we must be extremely careful because the match may be poor.

Another point regards the matter of handling grafts. Grafts are delicate, and they necrose easily. I would say that in handling them, one should use a small hook or forceps, grasping underneath in the subcutaneous tissue. Handle them extremely gently. Don't wash them with saline. Place them in the bed, and in the gentlest manner possible suture them by interrupted sutures.

Do not use catgut on grafts because it acts as a foreign body. Also, don't tie the knot on the graft. The graft has sufficient difficulty without this. Another point of importance is the condition of the bed. If it is extremely scarred and appears to have poor nourishment for the graft, you should select a type of graft that is sliding or rotating and that has its own blood supply, at least partially so. Do not use a free graft.

Another point which has been overlooked by individuals who are highly specialized is the condition of the patient. We sometimes become so interested in the surgery which we are going to perform that we forget to pay attention to the condition of the patient. He may be an old, undernourished, ill individual, and naturally we will not have the advantages that we will if we place the patient in the best possible state of health.

Scar tissue is important in the success of the graft. It seems to set the stage, and we must reckon with it before it goes through its stages of contraction and destroys the graft. In some cases in which there is heavy granulation, one can use roentgen therapy possibly a week before, apply the graft, and after the graft is healing, resume the therapy. That has, in some cases, reduced the amount of the scar formation and saved grafts which would otherwise be ruined.

We must be careful to remove all the scar tissue from the socket. That has been discussed before this evening. It seems, however, that if we leave scar tissue we may stand by helplessly when the prosthesis is extruded.

EDMUND B. SPAETH, M.D., Philadelphia: There are two or three facts I want to add to this discussion because I think that they are important. First, the closing sentence of Dr. Singer's paper is most significant. I will repeat it, "It is hoped that this review will help, by its reiteration of basic principles, our future success in grafting." Plastic surgery, gentlemen, is still the most poorly done procedure in ophthalmic surgery.

Next, I wish to call attention to this matter of "the types of graft" to be used in any one case. A case is doomed to failure or to indifferent success if a full-thickness graft is used when a split graft should have been used, or if a split graft is used when full-thickness skin was indicated, or perhaps most important, if one fails to use a pedicle flap when circumstances indicate it would serve best. Many of us had to learn this by experience. There is no reason why others must go through this same period of learning by trial-and-error methods.

The next point to emphasize is the matter of mucosal grafts and their application. Fre-

quently the mucosal graft is cut entirely too thick. Dr. Singer spoke of identifying the epithelial surface; this is important with a graft of the proper thickness. A silk suture for mobilization and handling of the graft also identifies the external surface. The next point relative to these grafts is important: one cannot—in fact, dare not—use mucous membrane (or conjunctiva) and skin together in the same case if they are in contiguity or continuity. If this is permitted, an intractable conjunctivitis or keratitis will occur which is most unpleasant and quite unnecessary. One should not use mucous membrane for any reason other than that of substitution for conjunctiva. When both epithelium and mucous membrane are necessary for an eyelid reconstruction or a coloboma repair, these two membranes must be "back to back" and never in juxtaposition.

Dr. Singer mentioned the immobilization of the graft. It is just as important to immobilize the graft recipient site. For that reason, it is wise to dress all grafts, full thickness or razor cut, with an overlying mold of dental stent. This mold, made of warmed dental stent, is formed over the recipient site before the graft is placed into position. After the graft is in position, this mold is bandaged down over the corrected site, thus immobilizing both the graft and the graft site.

If my discussion has added but little to Dr. Singer's presentation, it may have served to emphasize again a most important dictum relative to the use of mucous membrane. This is a most valuable graft when used properly, but it should not be misused.

CROWELL BEARD, M.D., San Jose, California: Dr. Singer's paper is most valuable in its emphasis on some of our causes of failure in graft work in ophthalmology. I believe that I have had my share of failures. A review of my kodachrome slides shows that I have failed to record them. A careful review of our past failures can do much in preventing future ones.

I believe that most of my poor results in grafting have resulted from a poor choice of donor site or from inadequate measures to prevent contraction. The ease with which buccal mucosa can be obtained often causes us to obtain a graft from the mouth of such size that it could easily have been obtained from the conjunctival fornix of the same or opposite eye. The amount of elastic tissue in buccal mucosa is greater than that in conjunctiva, and it tends to contract more. For this reason and because buccal mucosa tends to retain its pinkish color when grafted, a poor cosmetic effect may result when this tissue is used to replace bulbar conjunctiva. I have had some success with a composite graft of conjunctiva and buccal mucosa. Available conjunctiva is used to fill a defect on the globe, but buccal mucosa is used in the fornix and on the tarsal surface. I wonder whether better results might be obtained in many conditions if the tarsal surface were to be grafted with available mucosa and the bulbar surface were left bare to regenerate from the conjunctival edges. In a patient with annular epithelioma, the conjunctiva and cornea were excised about 8 to 10 mm. out from the limbus in all quadrants. The conjunctival edges were sutured to the bare sclera well out from the limbus, and satisfactory conjunctiva regenerated without deformity. The patient retained good vision through a central area of uninvolved cornea.

Romanes' use of homografts of skin in replacing conjunctiva is ingenious and interesting. I have had no experience with such a graft. If his cases were not followed by any attempt of the fornices to shrink, this procedure warrants extensive use.

In skin grafting, the source of donor site is especially important. The best results in texture and color match can be obtained by the use of lid skin to replace lid skin. An exception could be made of the lower lid. The lower lid does not serve as a good donor site in many cases because of the danger of ectropion. Many times upper lid skin is too thin and it may be darker. Here a rotating flap from the area over the zygoma or a free graft from the retroauricular area may be best. Dr. Singer has emphasized that supraclavicular grafts and split-thickness grafts from other areas are of limited usefulness. He has brought out that allowance must be made for late contraction if these sites are used.

Pigmentary changes in skin grafts are annoying. Colors can be matched to some extent by the tattoo technique of Brown and his colleagues. However, these have a marked tendency to unpredictable change from the original color tone applied. The thin skin of the lids is difficult to tattoo evenly. I have used tattooing with some success about the brow and eyelash area but not on the loose grafted skin of the lids. I should like to ask Dr. Singer whether he has utilized tattooing and, if so, what his degree of success has been.

I should like to complement Dr. Singer on his paper. I am sure we can all profit from it in reducing our percentage of poor results in grafting in ophthalmology.

Symposium: Foreign Bodies

INTRAOCULAR MAGNETIC FOREIGN BODIES

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THE principle factors involved in the extraction of metallic magnetic foreign bodies from the eye are four in number. Much of the success connected with the extraction of a magnetic metallic foreign body from the eye and with the conservation of the globe, as much as is possible for any given case, is directly related to the attention paid to these basic factors.

The four are (1) the magnetic quality and size of the foreign body; (2) the power and type of the magnet to be used; (3) the distance of the magnet from the foreign body (related to factor 1); and (4) the technique to be used, i.e., the utilization of an anterior route or of a posterior route for the extraction. The discussion of these four factors is the purpose of this portion of the symposium.

Magnetic Quality and Size of Foreign Object

The surgeon must adjust his technique to the circumstances present. There is no doubt that industry is using, increasingly and to a tremendous extent, various new alloys with poor magnetic qualities. At the present time the surgeon has no practical way of modifying the inherent magnetic quality of a foreign body in the eye. If a metallic foreign body of low magnetic quality is placed in a magnetic field, its magnetic quality will improve to a certain extent, but theoretical and actual magnetic saturation are reached very quickly. This is one reason why repeated applications of the magnet are not increasingly successful; in fact, they are unnecessary, and they generate undesirable heat in the magnet wiring. Because of this the pulling power of the magnet is actually decreased.

A careful history frequently will give the operator a good idea of the type of metal in the foreign body. It is not uncommon for the patient to be able to tell the exact source of the foreign body, e.g., "a scale of iron from the end of a cold chisel," "a fragment of a broken sewing machine needle," or "a chip of high carbon steel from a bar of stock in a milling machine." Thus, the accurate history will frequently help the doctor to determine whether a foreign body is magnetic or not.

Foreign bodies of low magnetic quality can be extracted only by increasing the power of the magnet and the size of the core-point in the magnetic field (the tip of the magnet), and by the approximation of the magnet tip to the foreign body. Most iron and steel foreign bodies, 10 mm. from the magnet point, can be extracted with ease with an ordinary magnet.

Extraction of particles made of alloys of low magnetism frequently requires actual contact, or a very close approximation, of the particle with the magnet point. Too often an operator will be

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working with the wrong type of magnet for the specific case, or working with the magnet at an improper distance for that specific magnet. The problems of these relationships can be considerably minimized by a good history, that is, knowledge of the magnetic and metallic qualities of the foreign body.

The size and shape of the foreign body are very definite factors in determining the technique to be used in magnetic extraction. They are probably the most important factors in deciding whether to use an anterior or a posterior route for the extraction. A foreign body which, because of its size or shape, could become incarcerated in the root of the iris or in the ciliary body or could damage the suspensory ligament while passing through it in the process of an anterior-route extraction should be removed by the posterior route. This principle cannot be questioned. Further comment will be made on this point in the discussion of technique. Roentgenograms showing anterior-posterior views, as well as lateral views, are usually necessary to demonstrate the shape of a foreign body.

Power and Type of the Magnet Used

One must remember that the hand magnet is essentially a contact instrument and loses its value very rapidly as the distance between magnet tip and foreign body becomes greater than the thickness of the cornea or of the sclera. Only the giant magnet permits varied manipulations at different angles for attracting and/or dislodging an incarcerated fragment and for guiding a metallic particle around the posterior surface of the lens and through the suspensory ligament into the posterior chamber of the eye.

Lancaster's criteria for good magnets are still correct, as they were when he first stated them:

> Unless a giant magnet will pull a tiny steel ball with a force of over 50 times its weight at a distance of 20 mm. and unless a hand magnet will pull a tiny steel ball in contact with its tip with a force of more than a thousand times its weight, neither are good magnets.

Hence it can be seen that a hand magnet cannot be used to replace a giant magnet when there is a demand for a diffuse magnetic field which permits a necessary variability.

The giant magnet has its maximum usefulness by reason of its size, shape, and mounting; in fact, great adaptability. It cannot be substituted for the hand magnet. It is true that a giant magnet of the Haab ring type would be ideal, theoretically, in answering the demands for both.

In my experience, the ideal situation is to have both giant and hand magnets available; both types are occasionally necessary. If I could have but one of the two, I would choose the giant magnet. Under such circumstances, however, very definite and positive changes in the technique for the use of the giant magnet as a substitute for the hand magnet must be made to save the eye from unnecessary damage. This is best illustrated in the following statement:

> The giant magnet would be ideal for removing a scale or chip of iron lying in the vitreous, first bringing it through the suspensory ligament into the posterior chamber behind the root of the iris. From there the hand magnet should be used to guide a foreign body either through the iris root or along the posterior surface of the iris and through the pupillary aperture and then into the angle in the anterior chamber.

Distance of the Magnet from the Foreign Body

The third of the basic factors is the distance of the magnet from the foreign body. This involves a consideration of certain principles connected with

magnetic fields in their application to the removal of foreign bodies. The power of the magnet to attract a magnetic foreign object diminishes relative to the increase in the distance between the magnet and the object. If the pulling power of the magnet at a distance of one millimeter is one gram, by increasing the distance to 2 mm. the pulling power becomes only one-eighth gram, and at 4 mm. it has dropped to one sixty-fourth of a gram, and so forth. Nearer than one millimeter the pulling power increases tremendously up to the maximum at actual contact. Figure 1 illustrates this.

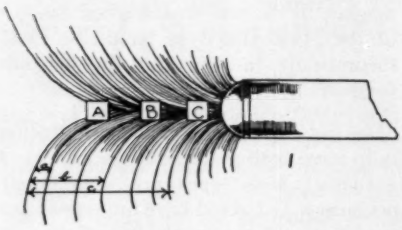

FIGURE 1.

When extracting a foreign body with a magnet, one must remember that all foreign bodies when magnetically attracted move with their longest axis parallel to the pull of the magnet, that is, in line with the core of the magnet, never at any great angle to it. Deviations from parallelism are only mechanical and are due to the friction of the various ocular structures with the foreign body during the extraction. One can see from figure 1, that foreign bodies A, B, C, as they lie in the field of the magnet, are exposed to decreasing amounts of magnetic force because the farther they are from the tip of the magnet, the less dense, hence less effective, is the magnetic field. The arrows show the relative amounts of effective magnetism for A, B, and C at their respective distances from the magnet. This same principle is illustrated in figure 2, which shows the increasing magnetic effect upon foreign bodies of various sizes, all at the same distance—A, being the smallest, B, next in size, and C, the largest; and the astonishing increase in the effective magnetic field under these circumstances.

Figures 1, 2 and 3 illustrate another point regarding magnetic fields. The most effective magnetic point is one with a blunt tip, about as broad as it is long

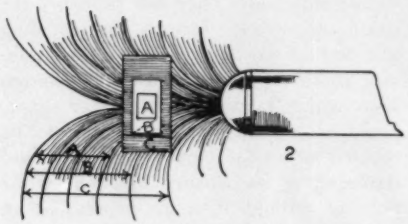

FIGURE 2.

and roughly pyramidal in shape, because the most powerful magnetic pull is exerted from the center of the point where the density of the metallic tip, a continuation of the core of the magnet, is the greatest. The magnetic lines of force which fail to reach the foreign body are wasted.

The weakest magnet point is the long, thin one (fig. 3). When this point is used at a distance of more than one millimeter from the foreign body, the magnet has lost practically all of its value. Such a magnet point is useful only for drawing a small ferrous foreign body

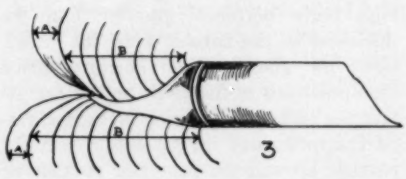

FIGURE 3.

out of the angle of the iris in the posterior chamber, through the pupillary aperture and from there into the anterior chamber, with minimal damage to the internal eye structures. This procedure should be evaluated in the light of the damage which could be done to an eye when this same manipulative procedure is attempted with a stronger magnet or with a giant magnet.

Figures 1, 2, and 3 emphasize the place in this surgical procedure for the various types of magnets available and the various magnet tips or points to be used. Usually it is useless to work with any type of hand-magnet head except the blunt, pyramidal-shaped tip with the broadest magnetic field. The introduction into the eye of forceps, probes, and similar instruments in contact with the magnet tip is of little value, as explained above, as far as magnetic attraction is concerned. This procedure may also cause considerable damage to the retina and/or the choroid.

Surgical Techniques

An important consideration in the surgical removal of magnetic foreign bodies is the question of anterior route versus posterior transscleral route. In general, the smaller the particle, the greater is the indication for the anterior route; larger particles should usually be removed by the posterior transscleral route. An intact lens should be preserved. In the presence of a damaged lens the anterior route is indicated not only for small particles but also, in some instances, for larger fragments. Foreign bodies that have entered through the posterior segment of the sclera or which lie in the posterior portion of the globe, should be extracted from the eye by the anterior route, all other factors permitting. If the point of entrance is well behind the ciliary zone and anterior to the equator, the extraction may be done through the point of entrance. The giant magnet can bring the foreign body toward this point of entrance for easier hand-magnet removal.

When the particle lies generally in the region of the equator, on or in the retina, and a posterior route of removal is deemed wise, the incision for the extraction should be made in the sclera as close as possible to the particle. The hand magnet should be used for the extraction. Sclerotomy incisions and points of entrance should be rimmed with diathermy punctures to guard against a later retinal separation.

Extraction of foreign bodies suspended in the vitreous usually requires use of the giant magnet. Early surgery is perhaps the greatest factor in these cases unless the foreign body lies wholly within the lens. When only a hand magnet is available, precise localization is absolutely necessary. With the giant magnet this is less important, though accurate information about the shape of the foreign body is of great importance.

Permanent magnets are of little value except for the removal of foreign bodies in the anterior chamber, and even for this purpose they are of doubtful value because their magnetic force is slight.

Foreign bodies which are incarcerated in the ciliary body must be removed through the anterior route from the anterior chamber angle. Foreign bodies of unusual shape, however, when adherent to the retina at or behind the ciliary body in the pars ciliaris of the retina, may be removed through a posterior route by means of a posterior sclerotomy (radial incision of the sclera with stay sutures in the sclera, as used for the sclerotomy of a cyclodialysis) behind the ora serrata. The magnet can move the foreign body away from the ciliary processes, thereby preventing entanglement in them, and then deliver the foreign body through the sclerotomy incision.

Foreign bodies in the posterior chamber or in the anterior chamber angle can be visualized with a contact glass, if necessary. An incision in the iris may be necessary; one made with a knife needle may be sufficient to permit removal of the particle from the posterior chamber through a keratome incision at the limbus by means of the hand magnet.

When metallic foreign bodies are removed from the anterior chamber, it is important to make the incision perpendicular to the cornea and not obliquely at the angle. The obliquely placed incision will form a shelf, making difficult the removal of a flat or scale-shaped particle. If iris prolapse results from an attempt to remove an incarcerated foreign body by a keratome incision and use of a magnet, a single meridional forceps-scissors iridotomy may permit removal of the particle through the iris and subsequent re-placement of the iris.

Foreign bodies which are known to be under the retina or incarcerated in the retina must be removed by a posterior route, with the incision in the sclera at the site nearest the object. Transvitreous delivery will almost certainly cause a sudden and abrupt separation of the retina because the pull of the extraction causes the retina to tear. This statement applies to removal of foreign bodies which, as seen with the ophthalmoscope, are buried, in part, in the retina.

Retinal separation is an ever present danger in posterior-route extractions even under the best of circumstances. Means to prevent this have been discussed; but deliberate damage to the retina resulting from erroneous choice of the anterior route for extraction of a foreign body should be avoided. These cases must be carefully studied to plan the direction of magnetic pull which will cause least damage to the retina. Stallard[1] feels very strongly that the bogey of retinal separation after foreign body extractions by the use of the posterior route is unwarranted. In fact he stated that if retinal detachment occurs as a sequel to removal of a foreign body, it is due to the nature of the penetrating wound and the resultant pathological changes within the eye rather than to the surgical intervention, if it has been properly planned and carefully executed.

When a giant magnet is available, a good working rule is to use it and to attempt an anterior-route extraction whenever any doubt exists as to which of the two procedures is the better for a given case. In general, the conditions which demand a posterior-route extraction are few, but when they are present they also are rather inflexible. The first statement may be debatable, however the second is not.

The path of entrance of a foreign body, its size, and its position are not the only points to be considered when discussing the choice of route for extraction of the foreign body. It would be quite absurd to jeopardize the entire eye by removing a huge intraocular foreign body, such as a section of steel wire cable, a half-inch long, by the anterior route. On the other hand, a small foreign body can be carried through the zonule around the equator of the lens, and into the anterior chamber from the posterior chamber without danger tc the lens capsule, except under most unusual circumstances.

The giant magnet is first applied to the sclera over the point at which the foreign body has been localized, with the axis of the magnet in line with the position of the foreign body and with the point of contact of the magnet tip on the sclera. The magnet tip is then slowly moved, still being held in close approximation to the sclera and at this same constant angle until the magnet point has passed the limbus and is over the anterior chamber angle. One should

now see a bulging of the iris as the foreign body enters the angle of the posterior chamber. The giant magnet is then lifted from the sclera, and discarded. The hand magnet is applied in contact with the cornea at a point slightly away from the foreign body, that is, with the magnet point in the center of the pupillary aperture. An attempt is now being made to move the foreign body into the pupillary aperture and on to the anterior surface of the iris and, at the same time, to prevent its incarceration in the meshes of the iris stroma on the posterior surface of the iris. If this result cannot be achieved very promptly, a keratome incision at that point should be made without delay; the iris should be incised with a knife needle, and the foreign body withdrawn through this opening in the iris. At times it may be necessary to do a complete peripheral iridectomy.

The removal of a magnetic foreign body through the posterior route using the giant magnet is undoubtedly a simple procedure. It is, however, fraught with danger because of the possibility of subsequent retinal separation. The conjunctiva is opened; the point on the sclera for the incision is rimmed with diathermy punctures; the sclera is incised meridionally at this point, as close as possible to the site localized by roentgenographic findings; the blunt, cone-shaped magnet tip is placed against the sclerotomy incision and the electric circuit is closed. Foreign bodies of irregular shape will be extracted with the smallest diameter presented first.

Following the posterior-route extraction of a foreign body, the surgeon should carefully close both sclera and conjunctiva. The immediate postoperative care should be the same, for several days, as that following surgery for retinal separation.

FOREIGN BODY LOCALIZERS

Nothing has been said herein relative to the use of magnetic foreign-body localizers. These have a definite place in the removal of intraocular foreign bodies. It is sufficient in this presentation to note the following significant factors in their use:

(1) The presence of iron or steel, if superficial and of considerable size, may be determined. (2) The presence and position of the foreign body is more easily determined if it has been previously magnetized. (3) The depth of the foreign body may be inferred from the degree of deflection of the needle. (4) A change in the position of the foreign body after the application of the magnet may be ascertained.

An acceptable localizer is indicated in instances of foreign bodies with doubtful magnetic qualities; in considering the question of double perforation; in deciding upon a possible or probable extrabulbar position for the foreign body; and for diagnosis in possible nonmagnetic foreign bodies when the first surgery had been unsuccessful, it being assumed that all factors connected with this surgery were properly carried out. Such a localizer is on the market; it has been at times, practically invaluable, though it is not routinely necessary.

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INTRAOCULAR NONMAGNETIC FOREIGN BODIES

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IT has been stated that a magnetic intraocular foreign body presents serious challenges to the surgeon. One might add that a nonmagnetic particle certainly presents more complex problems. Even in those cases in which the roentgen localization of a foreign body may not be quite exact, a magnetic splinter in the vitreous will be attracted toward the pole of the energized electromagnet. On the other hand, when the intraocular foreign body is nonmagnetic, the surgeon must know its precise localization; he must be able to see the particle in order to grasp it with forceps during surgical extraction.

The number of cases of nonmagnetic intraocular foreign body has increased in recent years due to the employment of various alloys and the spread of industry. Not all of the eyes involved in these cases must be lost. Modern ophthalmic surgery offers reasonable hope of sight preservation for many of them.

The problems of accurate diagnosis and the evaluation of damage done to an eye obviously affect the management of the case and the prognosis. In cases of penetration of the globe either by magnetic or nonmagnetic particles, the amount of anatomical and functional change must be determined. The extent of corneal, lenticular, uveal, retinal, scleral, and other types of damage must be ascertained. The extent of the impairment of visual acuity, visual field, and motility should be known.

The likelihood of infection must be considered and preparations made to combat such infection with sulfonamides, antibiotics, and other drugs. My personal clinical experience has definitely proved the importance of controlling severe traumatic and chemical inflammation by the administration of the adrenal steroids or corticotropin. Parenteral foreign protein therapy, either boiled milk administered intramuscularly in 5 to 10 cc. doses or typhoid vaccine administered intravenously, is also helpful in this regard.

It should be remembered that some foreign bodies, particularly those that come from the battle field or from the farm, may carry tetanus or gas bacilli. In such cases, one must be prepared to administer specific antiserums to protect the patient against the possible toxic effects of these organisms. Care must be taken to avoid serum reactions in hypersensitive patients.

The presence or likelihood of complications, such as uveal or vitreous prolapse, traumatic cataract, and retinal detachment, requires evaluation and resolute decision by the surgeon from the standpoint of therapy or prevention.

The chemical composition of the intraocular splinter should be known. Small chemically inactive particles, such as glass and coal, may often be left un-

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disturbed. On the other hand, chemically active foreign bodies, such as iron, copper, brass and wood, must be removed to avoid their destructive and toxic effects on the ocular tissues.

The above-mentioned items and problems of management, surgical techniques and other pertinent problems comprise the discussion at hand.

History

A meticulously compiled history is paramount in significance. This begins with an inquiry into the details of the accident, including date, time, place, and the manner of its occurrence. For medicolegal reasons, it is important to know whether the injury occurred at home or on the job. From the history, information may be gathered about the chemical composition and magnetic qualities of an intraocular splinter.

There are certain pertinent questions: Was the accident caused by striking a steel tool against a steel, iron, brass, copper, or stone object? Was the "material-shattering" work in the hands of the injured man or in the hands of a nearby worker? (The injured person may be a helper, foreman, onlooker, or passerby.)

Explosions which cause ocular injuries are the most frequent sources of single or multiple nonmagnetic foreign bodies of varied nature or composition.

The next question is, Was the mishap caused by firearms? Fragments of breechlock or gun barrel are usually magnetic (steel). Particles from exploding cartridge fragments are usually nonmagnetic (brass or copper). Shotgun-projectile injuries are caused by lead shot (nonmagnetic). Air-rifle injuries are usually caused by small copperclad magnetic steel pellets.

An examination of the tools employed, or of explosive particles lodged in the skin near the injured eye, may give a clue as to the nature of the intraocular fragment. Grit, sand, stone, lashes, wood splinters, and thorns are some of the substances, aside from metal or glass, which may enter the eye as the result of an explosion. Such accidents often result in multiple intraocular foreign bodies.

The management of the case depends upon the surgeon's knowledge of the circumstances surrounding the accident.

Eyes injured in accidents occurring in the barn, stable, or automobile shop are likely to be infected. All intraocular particles should be considered as potential bearers of infection.

The ocular history of the patient should also be ascertained, with reference to previous visual impairment or inequality, amblyopia, the wearing of glasses, previous ocular injury, disease, anomaly, strabismus, ocular treatment and surgery. Questioning should be indirect rather than leading. Present and past medical history may also be significant (diabetes, hypertension, head injury, neurologic disease, etc.).

Examination

The importance of a careful subjective and objective examination of the injured eye, of the fellow eye, and of both adnexal regions for evidence of trauma or disease becomes obvious. The visual acuity should, of course, be recorded in all instances. When determining the visual acuity, tests should be made first of the naked eye, then of the eye with a correcting lens, if worn, and then of the eye with a pinhole disc diaphragm, if indicated.

I recall several cases in which the chief complaint dealt with an injury to only one eye. Examination revealed a fresh injury of one eye and a retained intraocular foreign body or healed perforation in the fellow eye.

For a complete diagnosis, it may be necessary to use any or all of the diagnostic armamentaria at our command, including biomicroscopy, gonioscopy, and transillumination.

Examination may reveal perforations, foreign bodies, or other pathologic conditions of sclera, cornea, iris, lens, or retina. In addition to a foreign body, hemorrhage, exudate, or other tissue changes may be found in the anterior chamber and vitreous. There may be retinal and choroidal lesions. Siderosis or chalcosis may be present after retention for four weeks or longer of an intraocular splinter of iron or copper, respectively.

A perforation of the cornea, an iris hole, or trauma of the lens will leave permanent telltale evidence of recent or old injury. A small scleral perforation, when seen within a few hours after injury, usually has an associated subconjunctival hemorrhage. This is an important clue in locating the wound of entry. After a few days, such subconjunctival hemorrhage becomes absorbed, and a small sealed scleral hole may elude the most careful search. Larger scleral perforations usually gape and often show some vitreous or uveal prolapse. These require surgical closure.

Transillumination may facilitate the demonstration of iris holes and other lesions.

Gonioscopy is invaluable for locating foreign bodies in the anterior chamber angle. On occasion, such particles may be covered by exudate or organized connective tissue. Gonioscopic search is also very helpful in finding equatorial lens lesions as well as peripheral retinal lesions.

Perimetry may demonstrate a lesion of a bundle branch when the retina is damaged near the disc, or it may indicate an incipient retinal detachment following a traumatic retinal rent. When an eye is found to be soft on palpation or with a tonometer, the presence of a leaking perforation is indicated.

Records, diagrams, and photographs (when possible) are important in all cases of injury from the standpoint of diagnosis and management, and for many other obvious reasons.

ROENTGENOGRAPHY AND LOCALIZATION

After the eye has been surveyed, a careful roentgenographic examination is necessary in each suspected case of intraocular foreign body, including any case of uveitis or other inflammation of unexplained origin. It is wise to consult a roentgenologist who is interested in meticulous foreign-body localization and is familiar with a variety of localization techniques and the problems involved. He should have the guidance and cooperation of the ophthalmologist in each case. The exact position, size, and nature of the foreign body may thus be ascertained. Nonmetallic particles are not magnetic. They are seldom opaque to the x-ray.

There are a number of localization techniques applicable to the eye. All are subject to varying degrees of error, which must be recognized. Most techniques are based on the measurements of an assumed standard globe, which has an anteroposterior diameter of 24 mm. and a transverse diameter of 23.5 mm. It is obvious that variations in size of the eyeball do occur within a group of patients. Distortions are also introduced by technical factors. Some localization methods are subject to error because they do not preserve the desired constant relationship between (1) the artificial x-ray-opaque localization reference points, on or near the eyeball surface, (2) the anterior-posterior axis of the globe, (3) the central x-ray beam,

and (4) the photographic plate. Thus, a metal corneal localization ring, or the lead dots of a Comberg lens, should be parallel to the limbal plane. This plane should be parallel to the photographic plate. The central x-ray beam should coincide with the visual axis and should be perpendicular to the x-ray cassette and to the plane of the corneal limbus.[11]

The experienced technician can reduce the likelihood of localization errors by employing special methods and eliciting proper cooperation from the patient. The most frequently employed roentgenographic methods for foreign body demonstration and localization may be enumerated as follows:

1. Stereoscopic scout roentgenograms (posteroanterior, with patient's chin and nose touching the cassette)
2. Sweet's method of localization
3. Comberg's technique of localization, using a contact lens with four metallic limbal dots. (There are several modifications of this method.)[2,9,15,11]
4. Vogt's skeleton-free method (applicable to the anterior segment)[11,16]
5. Attachment of radiopaque rings or reference particles at indicated meridians on the eyeball surface[11]
6. Double exposure with the globe rotated in two opposite directions ("physiologic method")
7. Air injection in Tenon's space[5,10]

If there is any question about the presence of an orbital foreign body, it is wise to make stereoscopic scout roentgenograms. These should be taken in the posterior-anterior (P-A) position with the patient's chin and nose touching the cassette and his gaze maintained straight ahead, so that he constantly sees the reflection of his eyes in the polished surface of either the cassette or the x-ray table, or in a small thin accessory mirror set before his eyes. The chin-nose position is often called the Watters position. This position maintains a perpendicular coaxial relationship of the visual axis and the central x-ray to the photographic film. It throws the shadow of the petrous bone out of the orbit into the antrum and obviates obscuration of a minute foreign body by overlying bone.[11] When viewing these films in the stereoscope, one can visualize the presence and position of a foreign body in the orbit.

The Sweet localization method is well known and is reasonably accurate.

Comberg's technique of localization, using the contact lens with four lead dots, is very simple. I have devised a modification of this lens, in plastic with a small corneal vent hole and marginal scleral attachment holes, which has proved helpful.[15]* The vent hole prevents adherence of the lens to the freshly injured globe because of suction. The marginal holes permit attachment to the episclera with sutures. This keeps the lens from slipping on the globe. A chin-nose P-A view and a lateral view are obtained, with the patient's eyes fixing a point straight ahead. These suffice for localization measurements and plotting.

The skeleton-free[16,7,4] or soft tissue technique is used for small particles in the anterior segment lying just outside the bony orbit. It is performed with dental film held against the inner canthus[16] or in the inferior or superior conjunctival cul-de-sac.[7,4] Five exposures can be made on five separate films in any of the above target positions by having the patient look straight ahead, right, left, up and down. A larger portion of the anterior segment of the globe can be exposed temporarily to enhance the value

*Obtainable from Parson's Optical Co., 518 Powell St., San Francisco, and from The House of Vision Inc., 30 N. Michigan Blvd., Chicago 2.

of Vogt's method. This is accomplished by a retrobulbar injection of 4 to 6 cc. of plain normal saline solution or novocaine, one per cent.

There are many methods of attaching surface reference points. Goldmann's corneal or scleral ring may be employed. Small jeweler's rings can be attached to the globe with sutures. An improvised thin wire ring, 12 mm. in diameter may be attached to the limbus. In an emergency, two small lead shavings, or two broken-off complete eyes of suture needles, can be attached to the limbus at the 6 and 12 o'clock meridians. Any of the above devices serve as excellent reference points in the roentgenogram. With the patient's cooperation, roentgenograms are then made of the P-A view in chin-nose position and of a side view.

The "physiologic methods" of double exposure to show foreign body movement associated with globe rotation are subject to considerable error and are often misleading with reference to the extraocular or intraocular position of a particle. Therefore I do not rely on them.

Routine localization techniques based on the standard eyeball sometimes provide inconclusive evidence as to whether a particle is just inside or outside of the sclera. This presents a dilemma. The injection of 6 to 8 c.c. of air into the peribulbar Tenon's space, immediately beneath Tenon's capsule, to outline the globe helps to solve such problems. The injection is made between two adjoining rectus muscle tendons. This method was originally described by Gasteiger and Grauer,[5] and later by Spackman.[10] The injected air spreads through Tenon's space and thus outlines the globe wall. One or two pairs of stereoscopic roentgenograms are made as indicated in the particular case. Study of these films in the stereoscope gives one a tridimensional concept of the position of the foreign body. I have used this technique of air injection into Tenon's space in many borderline cases to establish or rule out a double perforation. This method has proved to be helpful, harmless, and nonirritating. Although I have used it many times in the past 20 years, it has never produced complications in my cases. The air in Tenon's space does not interfere with immediate or later surgery. All of the air escapes when Tenon's capsule is opened at operation. Infiltration of air into the conjunctival tissues must be avoided by holding the fascial puncture closed with forceps for 10 to 15 seconds. Emphysematous infiltration makes it impossible to outline the ocular wall properly and results in poor roentgenograms.

The roentgenologist prepares localization charts from the particular technique employed. The ophthalmologist should however study the original roentgenograms in addition to the prepared localization chart.

The Berman Locator[11,8,14,1,6]

The Berman locator is a very valuable instrument which depends on the electroinductive effects of a metallic foreign body on the electric field of the locator probe. It is of value in detecting magnetic particles and those nonmagnetic alloys which contain a sizeable percentage of iron or steel. It differentiates a magnetic from a nonmagnetic foreign body. It is extremely helpful in plotting the position of particles containing iron. It has not aided me in detecting, locating, or plotting either copper, brass, zinc, or other nonmagnetic, nonferrous particles; but it has provided invaluable help in the past 12 years in plotting the position of magnetic foreign bodies both before and during surgery.

Foreign Bodies in the Anterior Chamber and Their Removal with Forceps

A limbal incision, 6 to 8 mm. long, is required for the removal of deep cor-

neal foreign bodies which project into the anterior chamber. Preplaced sutures are helpful in closing this incision at the completion of the operation. The surgeon lifts the corneal flap and, if necessary, everts it to visualize the nonmagnetic particle. The foreign body is pried loose and then removed with a spoon or splinter forceps.[12] If the iris should prolapse, it is re-placed. The incision is closed with the above-mentioned sutures. The chamber is then restored with saline or air. A dressing is applied. The pupil should not be dilated until 24 hours after surgery.

Particles free in the chamber may be removed by the same technique. A splinter buried in the iris is removed by teasing the particle free with two forceps or by excising the tissue which contains it.

Gonioscopy is essential for actual visualization of foreign bodies in the anterior chamber angle. It is the only way one can be sure that a particle is in the angle. When only roentgenologic techniques are employed, one cannot be certain that the foreign body is precisely within the anterior chamber angle. It may be in the posterior chamber behind the iris, or in the ciliary body. The splinter may be glass, iron, copper, lead, stone, or of some other composition. It may be buried in exudate and may be the cause of recurrent ocular inflammation.

Various techniques have been suggested for the removal of foreign bodies within the anterior chamber angle. After trying most of them, I have come to rely on the approach from the scleral side of the limbus. One should avoid a corneal incision with the keratome because this presents an overlying corneal and scleral shelf which prevents proper access to the chamber angle. Even the Saemish-type peripheral incision with a narrow Graefe knife, which was formerly recommended, leaves much to be desired. The most adequate technique employs a flap-door incision in the sclera, two or three millimeters posterior to the limbus at the site of the foreign body. The anterior chamber is entered at the angle, and the particle is removed with forceps. This technique has been helpful in the removal of splinters which were situated in the anterior portion of the ciliary body and which protruded into the angle recess. After removal of the splinter, the scleral flap incision is closed with two 6-0 silk sutures. Atropine is not used until the first dressing period.

Foreign Bodies in the Posterior Chamber

Particles in the posterior chamber may be plotted by careful roentgen localization or by searching the space behind the iris diaphragm with the gonioscope through the dilated pupil. Before such a particle can be removed, it may be necessary to make a limbal incision and to evert the iris to expose the foreign body. The posterior iris tissue may be found to be discolored by chemical activity of the splinter. The particle may be covered by exudate. It is gently removed with forceps. When the particle lies between the ciliary body and the lens equator, the surgeon must resort to iridodialysis. I was forced to resort to this maneuver on two occasions. The iridodialysis was repaired in part by incarcerating the peripheral iris between the lips of the incision, which was closed with sutures.

Foreign Bodies in the Crystalline Lens

An intraocular foreign body in the crystalline lens may be left undisturbed until the lens becomes entirely cloudy. It is best to let the patient have phakic vision as long as possible. Unless the eye becomes irritated, surgery can be postponed until useful vision is impaired.

Then, the surgeon should perform an intracapsular extraction, removing the entire lens either with the erisophake or the capsule forceps, or by the Pagenstecher technique: placing a broad spatula behind the upper lens equator, breaking the zonule, and expressing the lens by pressure at the lower limbus. In young patients with an opaque lens and a subcapsular foreign body it has sometimes seemed wise to grasp the anterior capsule and subcapsular nonmagnetic particle with a toothed capsule forceps. The cataractous lens matter is then expressed and washed out with an irrigator.

Traumatic cataract is a serious complication of perforating injuries. If the capsular wound is small and closes over, the lens opacity may remain small and self-limited. A lens with an open capsule rent may swell rapidly and require early removal. If the tension is not elevated, it is best to postpone surgery until the entire lens is opaque.

Foreign Bodies in the Vitreous Chamber

Intraocular fragments of brass or copper produce enormous amounts of exudate. One can observe this process by ophthalmoscopy and in fundus photographs. It is an attempt by the organism to engulf and wall off the chemically active particle. The resultant exudate and the staining of the tissues by the metallic particle may prove helpful to the surgeon when he opens the globe. He may be able to trace the exudate to the site of the foreign body. Metal-stained tissue is often a clue to the proximity of the foreign body.

A few case reports indicate the procedure which I have followed.

CASE REPORTS

Case 1.

A 10-year-old boy had one eye destroyed by a dynamite-cap explosion. There was a piece of copper floating in the vitreous behind the lens. The foreign body was removed with direct ophthalmoscopic control.

Case 2.

The left eye of an 11-year-old boy harbored several intraocular foreign bodies for 18 months following a dynamite-cap explosion. Chalcosis of the crystalline lens was present. A sunflower-shaped tan-gray opacity was evident beneath the anterior capsule. Curiously enough, in spite of the large patch of discoloration of the pupillary portion of the lens capsule, the patient still had 20/30 vision. Management was conservative.

I am hesitant about attempting to extract multiple nonmagnetic intraocular foreign bodies because extensive damage to the eye is certain to occur during the procedure. An eye is usually sightless after such attempts have been made.

Case 3.

A 30-year-old exsoldier had been injured as the result of an explosion while he was in service. One eye was so badly multilated that it had to be removed. The other eye harbored several nonmagnetic intraocular foreign bodies as a result of the same explosion. I saw him first after he was discharged from service. At that time his vision was 20/100. He had chalcosis of the crystalline lens and of the peripheral corneal endothelium. His vision gradually deteriorated, and finally, his lens became completely opaque. An intracapsular cataract extraction was performed. Fundoscopy subsequently revealed retinal chalcosis and three foreign bodies in the vitreous, which were walled off by connective tissue. Ten years after the injury, he still had 20/30 vision with aphakic correction.

Nature tries to protect the eye against a foreign body by walling it off with exudate, which ultimately may be organized into connective tissue. However, excessive exudate may have a deleterious effect on the ocular tissues.

One should not operate immediately on every eye that harbors a piece of copper, particularly if the particle is not highly active chemically and there is no excessive exudation or endophthalmitis.

One may watch the eye and take all the measures to control infection and inflammation. There is usually time to do a careful localization and to come to a considered opinion and decision about proper procedure. Enucleation should be a matter of last resort only. Many gravely injured eyes can be saved, as is demonstrated by the following case.

Case 4.

The left eye of a 34-year-old man sustained a double perforation of the ciliary body with vitreous prolapse, traumatic cataract, endophthalmitis, and considerable exudate in the anterior chamber. There seemed to be no possibility of saving the eye. Enucleation was advised, but was refused by the patient. He agreed to antibiotic therapy and repair of the scleral perforations, however. He also accepted the responsibility for possible sympathetic ophthalmia. Repair was performed. The inflammatory process subsided ultimately. The traumatic cataract was later removed, and he now has 20/30 vision with aphakic correction. The patient took a chance, but he proved to be right.

TECHNIQUES FOR FOREIGN BODY EXTRACTION FROM THE VITREOUS

For many years, eyes harboring particles of copper, or of its alloys, in the vitreous were doomed to enucleation. However, in recent times, with newer techniques and instruments, in some of these cases the eyes have been saved. Several methods are available for foreign body extraction from the vitreous. The choice of method depends on the location of the particle in the eyeball, on its visibility with the ophthalmoscope, and on the transparency of the ocular media.

Method 1. Extraction Under Ophthalmoscopic Control

If the injured eye has a clear lens and a reasonably clear vitreous and the foreign body can be seen with the ophthalmoscope without excessive rotation of the eyeball, the method of removal of a particle from the vitreous with forceps under ophthalmoscopic control is applicable. This is usually done by direct ophthalmoscopy. After the usual preparation and the use of either local or general anesthesia, a retrobulbar injection of novocain with hyaluronidase is given to soften the eyeball. This reduces the amount and likelihood of vitreous loss after the globe is opened and extraction has begun. The point on the sclera nearest the foreign body is exposed through a conjunctival incision. This point is marked at the proper meridian with gentian violet. Generally, one seeks access to the interior of the eyeball through the region of the pars plana of the ciliary body in front of the ora serrata. When it is necessary to reach a foreign body in the retina, an incision directly overlying that site may be required. The region of the proposed flap-door incision (if posterior to the ora) is diathermized by application of the flat electrode using only sufficient blended cutting and coagulating current to make a gray, one-millimeter dot on the scleral surface. The flap-door incision is rectangular and is 3 mm. by 2 mm. or larger, depending on the size of the foreign body. After the scleral incision is made, silk sutures are placed in the lips of the wound so that it can be closed promptly after the foreign body is removed. Adrenalin, one per cent, is applied to the exposed uvea. The choroid and retina are then gently diathermized and opened with the point of a Graefe knife. The foreign body may appear in the wound as glistening metal, or it may be covered with exudate. If it is seen in the wound, it is grasped with a spoon-shaped forceps and extracted. The assistant should immediately close the scleral incision with the above-mentioned preplaced sutures.

If the foreign body is not seen in the wound, but can be visualized with the ophthalmoscope, it may be grasped with the tubular forceps which I have devised

for removing foreign bodies from the vitreous.[13] This forceps is constructed so that the blades do not move within the incision, a feature by which extensive damage to the neighboring tissues is obviated. Pressure on the eyeball by instruments and eyelids must be avoided while one observes the foreign body and the forceps blades through the ophthalmoscope. A relatively rapid movement in the vitreous will push the foreign body aside so that it evades the closing forceps. The approach with the forceps and the closure of the forceps blades must be slow and deliberate. Repeated reentry through the scleral incision is unnecessary and should be avoided. When the foreign body is extracted, the scleral wound is closed with sutures as in the previous instance.

Method 2. Transposition of Ophthalmoscopic Observations Onto the Sclera

In some cases the particle is attached to the wall of the globe. In such instances, the placement of the scleral incision may be determined by several methods:

a. The position of the foreign body, as determined by perimetric sighting with the ophthalmoscope, is indicated on the sclera.

b. A surface coagulation is placed in the vicinity of the foreign body, and the relationship of the surface coagulation to the foreign body in the retina is sighted. Two other coagulations then are placed so as to encompass the foreign body within the triangle formed by the three points.

c. A micropin stylus is placed through the sclera and retina at or near the site of the foreign body, and its relation to the foreign body is visualized with the ophthalmoscope, as advised by Atkinson and others.

These methods are applicable particularly when the foreign body is in the peripheral retina and when extraction under ophthalmoscopic control would be difficult. In any event, they help to place the scleral incision for the removal of nonmagnetic particles near the retina. In this type of case, it is sometimes necessary to detach a rectus muscle temporarily, for the period of the operation. A flap-door scleral incision is made after the scleral wall has been diathermized to prevent retinal detachment. The flap is turned back, and the area is inspected for discoloration of the choroid. This is usually found in the neighborhood of chemically active particles, such as brass or copper. If discoloration is found, the choroid and retina are treated with adrenalin (1:100) and diathermized; then an opening is gently made. If the foreign body is seen, it may then be removed under direct vision. If it is not seen at this point, a transilluminator applied near the central portion of the cornea may help to visualize it. In other instances, a headlamp worn by the surgeon may prove helpful in this type of case. If the incision is small and if preplaced sutures have been used, the loss of vitreous will not be great. The loss is minimized if the globe has been decompressed by the use of retrobulbar injection of novocain and hyaluronidase and if the patient has received diamox, 500 mg., two hours before surgery. After the incision is closed, it is wise to diathermize any visible recoil point on the retina, a point where the foreign body had originally struck the retina and bounced off to another place. In all such instances, only a minimal amount of current should be used on a dry eyeball, and only partially penetrating micropins used with the blended cutting and coagulating current. Detached muscles are now reattached with mildly chromicized catgut, and the conjunctiva is closed with a running suture.

Method 3. Transillumination

If the crystalline lens is cataractous or if the foreign body is near the wall of the globe or inaccessible to ophthalmoscopic observation, it becomes necessary to plot its location by other methods. This may be done by roentgenograms with a small jeweler's ring attached to the globe surface, or by transillumination through the pupil against the foreign body. The latter method casts a shadow of a foreign body near the sclera onto its wall. One may thus spot the proper position for the incision. The transilluminator, if brilliant enough, can be used either at the pupil orifice or at the wall of the globe opposite to the position of the foreign body. If the particle is small and is more than 2 or 3 mm. from the wall of the globe, it does not cast a shadow. If it is at the wall of the globe, or if it is relatively large, a fairly sharp shadow is cast. If the foreign body is a short distance from the globe wall, moving the transilluminator from side to side causes a parallactic displacement of the shadow. This maneuver must be performed in a darkened operating room.

Transillumination is especially helpful because one may be deceived by roentgen localization done with a patient in the prone position, whereas the operation is performed with the patient in the supine position. It is conceivable that gravity might change the position of the foreign body, so that at the time of surgery, the particle has migrated to a position other than the one originally plotted.

After the transilluminator has outlined the position of the foreign body, the eyeball may be opened in a manner similar to that in method 1. The foreign body may be visualized either with a headlamp or with a transilluminator and grasped with forceps. Standard closure is performed with preplaced sutures.

Method 4. Ophthalmic Endoscopy[1]

If the lens is opaque and the vitreous is clear, extraction by direct ophthalmoscopic control or by the transilluminator technique is not feasible. One must resort to the ophthalmic endoscope. Usually one or two rectus muscles should be detached. An incision 8 mm. long and 3 mm. wide, of the flap-door type, is prepared in the region anterior to the ora serrata, if that is feasible. The surgeon, having previously acquainted himself thoroughly with the use of this monocular instrument by dry surgical practice, now checks the instrument for performance. The diathermy having been performed, sutures having been preplaced, and the choroid having been coagulated, the choroid and retina are opened and operating room lights are dimmed.

The lamp of the endoscope is turned on, and the operating end of the instrument is dipped in the wetting agent, phemerol or 1:5000 zephiran. This precludes the formation of Newton's rings and other visual hindrances when the telescope lens touches the vitreous surface. The assistant must be careful to hold on to the endoscope sheath so that the surgeon does not accidentally thrust the instrument into the eye. The telescope and illuminating lamp, with a closed forceps in front of them, are brought in contact with the vitreous between the scleral lips of the incision. The surgeon's eye is at the eyepiece of the endoscope. His left hand supports the instrument. His right hand actuates the foreign body forceps. He looks into the vitreous through the inverted galilean telescope of the endoscope and pushes the forceps, which has been preset for a definite depth, toward the foreign body, which he visualizes. He then slowly opens the blades of the tubular forceps and gradually approaches the

foreign body with the forceps. This is the only part of the instrument which should penetrate the vitreous.

When the foreign body is grasped by the forceps, the surgeon keeps it in view while he pulls the forceps slowly up to the telescope. Having extracted the foreign body, he removes the instrument from the field of operation. The assistant closes the incision with preplaced sutures. Additional interrupted sutures are placed as required to close the scleral incision. As a rule, only a small amount of vitreous is lost during the procedure. The endoscope can be used only when the vitreous is clear. It is not helpful in a cloudy or bloody vitreous.

At the end of the operation a small amount of air is injected as an additional precaution against retinal detachment.

When the method of direct ophthalmoscopic control is not applicable, the globe may be further softened by paracentesis of the anterior chamber. This applies particularly to the techniques using the transilluminator and the endoscope, as described above. The paracentesis is performed just prior to the chorioretinal incision.

The following precautions should be observed while the endoscope is being used: Be careful not to thrust the instrument into the eye. The assistant must keep the terminal guard ring of the instrument in sight at the outer surface of the sclera. Only the end of the telescope, which is the central tube of the instrument, and the electric bulb should come in contact with the vitreous; and they should not be thrust into it. The choroid and retina must be incised to expose the vitreous. The small electric bulb must not be overheated, as heat may coagulate and cloud the vitreous. The proper rheostat setting should be determined by preliminary trial. Do not use house current through a transformer, as there is some danger of electric shock to the patient. Remember to dip the end of the instrument in the wetting solution so that the vitreous surface will not smear the objective lens of the endoscope. The illuminating lamp of the endoscope should be round, and its end must not be fused. A lamp with a fused end casts shadows. Only the forceps of the endoscope may be thrust into the vitreous, and this only to a predetermined and preset depth.

Method 5. Biplane Fluoroscopy

Tertz, abroad, and Cross, in this country, have reported success in the removal of nonmagnetic intraocular foreign bodies with the aid of the biplane fluoroscope. The author has had no experience with this method.

Method 6. Use of a Meta-magnet

There has been a moderate amount of publicity on the use of a meta-magnet for the extraction of nonmagnetic foreign bodies from the vitreous. This device has not yet been proved feasible for, or applicable to, the extraction of small foreign bodies from the eye. Furthermore, it is reported that it induces overheating of the tissues. The meta-magnet will probably be improved. At the present time, it does not have clinical application in ophthalmology.

DELAYED VISUAL IMPAIRMENT

At times, following a successful operation with an immediate good visual result, visual loss develops as a result of an unseen retinal tear and subsequent trauma.

Case 5.

Boy, age 12. Brass splinter from exploded cartridge in injured left eye. There was considerable exudate on the retina in the region of the foreign body. ACTH was administered parenterally to reduce inflammation. The particle was removed from the vitreous, and

subsequent vision, much to our surprise, was 20/20. The region of surgery had been diathermized and the retina appeared to be in good position. Examination some time after surgery revealed a tear in the retina at quite a distance from the site of the foreign body. This was apparently a recoil point. It was covered by exudate at preoperative examination and was not suspected. The parents would not agree to a second operation for closing the retinal tear. The youngster had had good vision for two years when he received a blow on the operated eye by a baseball. He was brought to my office a month later with an extensive detachment of the retina and an additional tear. In spite of two surgical interventions it was not possible to reduce the detachment or to get good vision in that eye again.

On the other hand, a case with apparent poor prognosis may turn out reasonably well.

Case 6.

Man, age 38. Injured at work. A piece of brass, irregularly shaped and measuring about 2 mm. by 1 mm., entered through the cornea, passed through the lens, and lodged in the vitreous, causing an endophthalmitis. This patient came from a distant city where enucleation had been advised. The use of intravenous ACTH by the slow drip method reduced the inflammatory reaction, so that this patient could be operated on two days after admission to the hospital. The foreign body was removed with the endoscope. The posterior perforation rosette of the cataract subsided. The lens opacity has not increased in the two years since the operation, although it is my experience that in cases in which the foreign body has passed through the crystalline lens the lens opacity usually increases. Vision has been maintained at 20/40.

In the majority of cases of nonmagnetic ocular foreign bodies, good vision is not preserved, but it can be in some cases. It is worth while to develop the various techniques so that when the problem arises, one may proceed with a definite plan of attack. The operation should be done only after the surgeon has prepared an adequate armamentarium and has acquired complete familiarity with his instruments. A trained assistant and a trained instrument nurse are essential, and every instrument must be at hand for the critical time when it might be needed.

CONCLUSIONS

1. A careful examination should be made and a complete history taken. From the history, one might determine the nature of the intraocular foreign body with reference to its chemical composition and magnetic state.

2. Definite knowledge about the extent of trauma to the various structures and about the extent of visual impairment must be acquired.

3. In every case, infection must be considered as a potential. Prophylactic and therapeutic measures must be taken.

4. Roentgenographic examination with various types of exposures, positions, and techniques should be made in each suspected case as indicated. Stereoscopic roentgenograms are invaluable, and accuracy in localization is paramount.

5. Removal of chemically inert foreign bodies should be avoided unless they are harmful because of their size or mechanical irritation.

6. Mutilating surgery and groping in the vitreous should be avoided. These procedures may result in destruction of vision or of the eyeball.

7. Retinal detachments should be anticipated, and when they occur they should be treated immediately. Precautionary measures against detachment should be taken in preoperative and postoperative care as well as during surgery.

8. Intraocular foreign bodies should be removed promptly to prevent their becoming enmeshed in exudate and organized scar tissue, and also to prevent the damage that results from their prolonged stay in the eyeball. The proce-

dure must be done only after adequate study of the case.

9. Conservative therapy in cases of multiple intraocular foreign bodies should be the prime rule.

10. A definite, well-considered plan for each operation should be formulated.

11. The surgeon should be thoroughly familiar with the surgical instruments at hand. He must be sure that they are in first-class working order.

12. The ophthalmic endoscope may be invaluable in appraising the damage to an eye from a retained nonmagnetic foreign body. The surgeon must be adept with the instrument.

13. Scleral and corneal incisions and wounds heal faster and better when they have been properly approximated with sutures.

14. The topical application of epinephrine, one per cent, and thromboplastin (or thrombin) to the surface of the ciliary body or choroid will sometimes prevent destructive hemorrhage. It is important to perform mild electrocoagulation of the exposed uvea before making the incision.

15. Enucleation is sanctioned only when the globe is collapsed and extensive vitreous loss has taken place and light perception is lost. When both eyes are injured, enucleation should not be done precipitately.

16. Foci of infection and other systemic conditions that may delay healing and cause complications should not be overlooked.

17. The danger of sympathetic ophthalmia should be recognized. Treatment of intraocular foreign bodies can safely be delayed no longer than three weeks after the injury.

18. The prognosis must be guarded in all cases of intraocular foreign body because of the impossibility of a precise evaluation of the trauma and of the variety of complications that may attend the injured eye.

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EXTRAOCULAR FOREIGN BODIES

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EXTRAOCULAR foreign bodies may be on or under the eyelids or within the lid structures, the cul-de-sacs, the cornea, and the bulbar conjunctiva. These foreign bodies include cement, cinders, emery, fish scales, pieces of wood, fish hooks, linseed, eye stones, and chemical particles from smoke. Some of my recent cases were those of patients who had a softened, denuded cornea. Working in railroad yards between chemical plants and steel mills, they had gotten smoke in their eyes. There was no history of any foreign body striking them.

Speaking of foreign bodies, one of the smallest ones almost evaded me. A man came to see me who had had inflamed eyes for two or three days and could not get relief. I looked at his eyes with the ophthalmoscope; there was a thin ring in the center of one cornea. I then looked at it with a loupe and discovered a fish scale on the cornea. I removed the fish scale, and that gave him immediate relief. That foreign body could easily have been overlooked.

One of the worst cases of extraocular foreign body that I have cared for was the following: The patient had moved his safety goggles up on his forehead. Drops of perspiration fell into the molten metal with which he was working, showering small casts of metal over both corneas and scleras. I first saw him when he was on the operating table, under anesthesia, for removal of the casts. After the casts were removed, I looked down on what were apparently two perfect eyes. The corneas were clear, but the scleras had a peculiar china-white appearance. Both of the scleras had been entirely coagulated by the hot metal. Four days later, I took out both eye balls, which had sloughed.

It is not within the scope of this discussion to take up the incidence of extraocular foreign bodies in comparison with the incidence of intraocular foreign bodies or other accidents. I believe that the extraocular far outnumber the intraocular, and the former may produce fatal or tragic results just as do the latter.

Most important is the care of the eye following the removal of these extraocular foreign bodies. If they are not properly cared for, simple foreign bodies may cause as severe disability, with loss of vision and loss of time, as intraocular foreign bodies. Still their prevention and removal and the aftercare of the eye receive little attention. Foreign bodies on the cornea are of as much importance as intraocular foreign bodies with respect to loss of vision and loss of time.

Of first importance in the consideration of ocular foreign bodies is prevention. The "safety man" has accomplished much in prevention and is a most important factor in this type of case. This subject has been ably treated in other discussions.

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For years, there have been numerous cases every day of foreign bodies on the cornea. These cases are of as much importance as the cases of intraocular foreign bodies. In some shops there was one employee to whom employees with foreign bodies were referred. This employee had a pocket knife with a sharp blade, and he removed the foreign bodies without the benefit of anesthesia. I remember that when I started out doing eye accident work in an industrial plant the employees said, "Oh, they must all go to So-and-So to have foreign bodies removed." The person who removed the foreign bodies was another employee. Some of the men preferred to go to this employee rather than to me. He would place the patient on a bench, take out his pocket knife, and remove the foreign body from the eye without administering an anesthetic.

A valuable asset in the treatment of these cases is the establishment of a first-aid station or dispensary conducted by a trained nurse or physician who can at least recognize the type of case that should be referred to the oculist. (I have had first-aid dispensaries under capable nurses or full-time physicians.) Some cases are referred to a hospital dispensary. Interns and residents should be trained in recognizing the seriousness of these cases and in the technique of caring for the simpler cases, for many times their mistreatment results in more serious conditions. I have been surprised at the lack of training that some of our residents and interns have had in eye work, and I have had the responsibility of giving them further training.

Now a little ancient history. I remember an old physician who called me in shortly before his demise. My preceptor had operated on him for removal of cataracts. While I was calling on him he showed me an eyestone. I wonder how many of you have ever seen an eyestone. A small, flat, smooth stone, picked up somewhere in a stream—it was used when a person had a foreign body in the eye. It was introduced under the eyelid, the idea being that the movement of the stone would displace the foreign body. Flax seeds were used in the same way. Another favorite method was to introduce a horsehair under the lid in an effort to take off the foreign body. You have also heard about the use of the tip of the tongue for this purpose.

Foreign bodies, loose on the conjunctiva or the lid or in the cul-de-sac, may be removed with a cotton-wound applicator. Removal of foreign bodies from the cornea is a different matter. I do not advise removal of foreign bodies from the cornea with a cotton-wound applicator. Most of these foreign bodies, whether from the street or the shop, are small particles of cinder. One side is smooth; the other side has teeth. An applicator wound with cotton, used in an effort to remove the cinder as it lies on the cornea, can take off the superficial layer of the cornea. I think that the proper procedure is to anesthetize the cornea and then lift off the foreign body with a sharp knife. Some of the most painful cases that I see in the office are those in which a cotton-wound applicator has been wiped across the cornea, removing the superficial layer. (I give my industrial nurses credit for avoiding such results.)

When a foreign body has been removed with a dry or moist applicator and a large or small portion of the superficial layer of the cornea has been removed also, the patient suffers a great deal of pain. In many cases the foreign body still remains on the cornea. With a sharp-pointed blade the bit of cinder can be lifted off with no damage to the cornea and the small abrasion beneath the foreign body causes no discomfort.

A great many cases seen by the oculist show what is erroneously called a "rust

ring." The majority of the foreign bodies that strike the cornea of the eye come from grinding wheels, and when they strike the eye they are intensely hot. The particle strikes the tissues of the eye and causes a third-degree burn. Often there is a slight stinging pain. The heat anesthetizes the tissue, and unless the foreign body is large enough to project above the surface of the cornea, the patient is unaware of its presence for two or three hours. Then the nerve endings recover sufficiently, and he is aware of something in his eye. Perhaps this occurs after he has gone home and then he seeks assistance.

What has happened? The hot foreign body has burned into the surface of the cornea. Examination shows a protruding black or brown foreign body which pushes against the upper lid and on the surface of the cornea causing pain. This particle from the grinding wheel, either emery or oxidized metal, is quite easily knocked off by an applicator in the hands of the nurse or physician; but after the local anesthetic wears off, the pain is as bad or worse than before. The presence of a rust ring is now discovered. (It is not rust, unless any oxidized tissue is termed rust.) When the hot particle struck the eye it burned the underlying tissue into a brown or black crisp scale, slightly larger than the foreign body.

If the heat was intense, a white ring of coagulated tissue may be seen surrounding the brown tissue or scale. This white ring is often mistaken for a collection of pus, but it is more likely to be sterile, white, coagulated corneal tissue. Many times such a lesion is reported as an infected ulcer when it is actually coagulated tissue around the former site of a foreign body.

These foreign bodies with the white ring usually are easily removable with a knife, as there is a line of demarcation around them. The patient with this type of foreign body presents himself complaining of severe pain, the pain having been intensified by the efforts of the nurse, friend, or doctor to remove the foreign body. Moreover, the superficial layer of the cornea has been removed. Following the removal of the original foreign body the wound is not painful, but as the anesthetic wears off, the tissues in the wound swell against the lid causing more pain, especially if the brown ring remains in the wound. It is now necessary to remove the rust ring to relieve the pressure on the wound. If the first operator has removed some of the superficial layer of the cornea, the pain is severe and an anesthetic must be given the patient.

As to the choice of an anesthetic agent, each operator has his favorite, so I will not dictate. Cocaine, 4 per cent, was formerly used. Its use has been abandoned because it dilated the pupil and softened the tissues of the cornea. A surprising number of patients ask if the surgeon is going to use an anesthetic which will dilate their pupils and interfere with their vision so they cannot drive home. For a long time I have used metycaine hydrochloride, 4 per cent, and found it most satisfactory.

Another subject, about which I have been criticized many times, is the use of dressings. Over a period of forty years I have used no dressings on eyes from which a foreign body has been removed; that is, when the foreign body only, not together with the superficial layer of the cornea, has been removed. An industrial nurse in our city remarked to me that when she went into a new plant she looked down the row of machines and was surprised by the number of white-bandaged eyes there were among the men. It was not one of my shops. In my industrial experience I have had one instance of infection among the cases in which I have used no dressings, and that

was due to a foreign body that was removed by a person who was not a physician.

If the cornea is denuded or abraded or if cauterization has been done, a dressing must be used. In order to care for a patient without using a dressing, the foreign body must be removed properly, then the patient is referred back to his shop dispensary where the nurse will give him hot compresses and possibly a drop or two of anesthetic agent during the day. He is seen the next morning if necessary. If, in the surgeon's judgment, the patient cannot be trusted to keep his hand away from his eye, or if a cauterization has been necessary, or if the patient will not have proper care until the following morning, a dressing should be applied; and the patient should be told that he must let it remain there. In a great many cases in which a dressing has been used, the patient takes it off as soon as he is outside the shop. If the patient goes directly home from my office, I order metycaine and merthiolate ointment and give him instructions; the patient reports the following morning.

Why use a dressing? The patient is more comfortable, particularly if he returns to work, if he has no dressing. A moist dressing over his eye will promote the growth of organisms.

Each operator has his own favorite instrument with which he is adept. The instrument-supply companies and hospital dispensaries furnish various types. One that I have seen in a great many dispensaries has a rounded, blunt end. One with a sharp diamond point can be used, but frequently it is dulled and so is ineffective in removing the brown ring. Some surgeons prefer a dental bur, the idea being that by rotating the bur the brown stain can be removed. I have used the bur, and in my opinion, it is an awkward instrument; it looks immense under the magnifier. Then there is the corneal curet. It is satisfactory if it is sharp and if the operator knows how to manage it; but it has to be sharp.

My preference is a sharp-pointed Graefe knife, at least 3 mm. long and with a very sharp point. If the knife can be inserted under the skin of your thumb painlessly, it is all right to use it; if not, it is not the proper instrument. With this knife the brown-stained tissue may be carefully dissected out. It may be adherent at one edge, but careful dissection will release it. Occasionally the rust ring breaks up into small segments. In this case a trick I learned from our friend Dr. A. D. Ruedemann works nicely. Wipe out the wound with an applicator on which there is a 2 per cent solution of silver nitrate. The next day the fragments will all be gone, or they can be removed easily. The thin brown-stained layer of tissue in the bottom of the wound need not be removed. It will gradually soften and disappear.

For removing foreign bodies, a good light is essential. One should be provided at the plant dispensary. There are a number of special lights on the market, but they are not needed in a dispensary. A 75-watt lamp with a good condenser on it provides all the light that is needed. Snell has devised his own electric eye spud and finds it very useful. I have not tried it, and I am too old to learn new tricks. I like my own.

Magnifiers are necessary and should be provided according to the choice of the operator. Some surgeons like one type; some like another. I use the old Cardington loupe, 10 power, and I think I have had great success with it.

Fluorescein solution is used by some surgeons to stain the cornea in order to ascertain the extent of a corneal wound. When fluorescein is used, no mercurials should be introduced in the eye, because a dense brown stain covering the wound and remaining for several days will result.

BACTERIOLOGY AND VIROLOGY OF ACUTE INFECTIONS OF THE RESPIRATORY TRACT

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BY INVITATION

AN attempt to categorize and discuss each and every agent involved, at one time or another, as an incitant of acute respiratory infection would be a Herculean task. The time required for such an endeavor would be prohibitive. Fortunately, common denominators transcend many species and even many genera of infectious agents. In addition to this, certain fundamental knowledge is necessary for the intelligent approach to and skillful handling of problems pertaining to infectious disease. Accordingly, one might predict that this presentation will be composed only of a series of isolated statements about pathogenic micro-organisms. Too often, however, the importance of the axiom in basic science is lost or weakened for want of application and illustration. This is true not only for the academic student of medicine but for every practicing physician, for every physician worthy of the profession remains a student for life. Therefore, I propose to discuss certain fundamentals with broad applications but to illustrate them specifically.

Micro-organisms exist as populations. This first axiom sounds trite indeed. It is a well-known fact among microbiologists, yet it is often overlooked, forgotten or ignored. This is an unfortunate situation, because so many of the frustrations which have occurred in the treatment of, immunization against, or unpredicted encounters with, pathogenic microbes can be explained by this fundamental truth. If we take a few moments to delve into the subject of microbial populations, we will see how important this subject really is.

As a starting point, consider the microbial flora of the oral and upper respiratory tract. A list of all of the microorganisms that have been encountered in these areas reads like a registry of almost all the organisms the sophomore medical student will encounter during his entire course in bacteriology, and it will include some that would not be covered. Some are pathogens in their own right; some are potential pathogens waiting for an opportunity to get beyond the confines of the mucosal barrier, or for man to upset a natural balance, which will give them the necessary advantage to propagate beyond their normal limitations.

To visualize this seething microbial population in number and kind is to see only one part of the picture. The interchange of force and function among these numerous varieties of life must be tremendous. As a first example one could cite antibiosis. This is a broad subject, but I shall illustrate it with only a few examples. If one made cultures from the throats of many people, he would expect to find only a small incidence of Hemophilus influenzae. If this experiment is repeated using the method of the late Sir Alexander Fleming,[4] that is, adding penicillin to the culture plates, one would expect to find about 85 per cent of the cultures positive for this same organism. By inhibiting the growth of gram-positive organisms, which are

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the normal antagonists of Hemophilus influenzae, the true incidence of this organism becomes apparent. Upon numerous occasions at the State University of Iowa we have followed mixed infections by culture, only to have the number of participating species diminish, or be reduced to one, with the appearance of Pseudomonas aeruginosa in the exudate. This organism is known to produce a potent antibiotic, unfortunately too toxic to use therapeutically but, in normal concentrations, effective against many of its fellow microbes.

Illustrations need not stop with bacteria versus bacteria. Bacteriological products, for example, polysaccharides, are known to inhibit or block viruses.[8] This is not to say that they combat the virus once the virus has penetrated the susceptible host cell but rather that they block the virus so that it cannot attach to the host cell. This phenomenon could occur in a mixed viral and bacterial population.

No doubt, at this point one feels it is well that micro-organisms are so considerate of mankind's welfare that they fight one another. Unfortunately, their purpose seems unpredictable, for synergism and symbiosis are also well known among many microbes. Mutual antagonism may be upset to the extent that almost pure cultures of some of the pathogenic species may replace the normal flora following the use of antibiotics. The upset of this balance fails to mean much until one recalls the tremendous reproduction rate of many micro-organisms. Some bacteria have been observed to reproduce themselves in nine minutes.[6] If the geometric increase of a single cell continues for twenty-four hours at this rate, the number becomes staggering. Multiply this number by the number of cells of the same species in a given locality and the product is astronomical. Need there then be any mystery why antibiotic resistance may develop overnight? Viruses also may have high multiplication rates and have been known to increase 1,000,000-fold in twenty-four hours.

All of the above phenomena need have little consequence if all individual microbial cells within a given species are identical. The mixed population concept extends beyond the consideration of mixed divisions of fungi, viruses and bacteria and beyond mixed species within a given genus. It must be considered even at the species and strain level. It can be truthfully said that there probably is no infection caused by a pure culture. The variation within a so-called pure culture is known to be considerable. Here again the illustrations could be many, but we will concern ourselves with only a few.

Adaptive enzymes known to occur among bacteria make a good example. Pure cultures tested on a substrate containing no lactose, for example, produce no lactase. The same culture grown in the presence of lactose succeeds in elaborating the enzyme lactase. The exposure of a pure culture of Neisseria to streptomycin has resulted in three major variants[11]: some cells were susceptible, others were resistant, and some were dependent upon this antibiotic. Hotchkiss[9] has been able to show a double adaptive potentiality on the part of Diplococcus pneumoniae. He was able to develop an antibiotic-resistant strain with a change of antigenic type. Both of these adaptations had been demonstrated before, but he showed that they could occur simultaneously. The importance of this observation is realized when one recognizes that such an accomplishment affords the organism two means of escape: one from antibiotic therapy and one from immunity.

All of the problems related to change in micro-organisms are not limited to

the practicing physician. In the field of diagnostic bacteriology may be found not a few. An example of change in the virulence of Mycobacterium tuberculosis following streptomycin and isoniazid therapy follows: A guinea pig was inoculated with sputum from a treated case of human tuberculosis. A small local node six weeks later was the only result. Was it, or was it not, tuberculosis? When this node was passed into a second pig, the second pig developed typical tuberculosis with liver and spleen involvement. The question could be raised as to whether or not these "attenuated" strains might not become virulent upon passage from human to human. Examples of antigenic change among viruses have also been established. Hirst,[7] Burnet and Lind,[2] and Gerber and his associates[5] have shown a multiplicity of antigenic varieties arising from what in the past has been considered uniform virus seed. So complete are some of these changes that immunity against parent strains becomes ineffective against the variants.

No one would be so impractical as to deny the phenomenal gains against infection resulting from antibiotics placed in the hands of the physician. The development of resistance to antibiotics by some bacteria and the deaths resulting from over-growth of resistant microbes following the use of antibiotics certainly do not contraindicate their use. However, promiscuous use of antibiotics against an adversary as versatile and wily as those we have mentioned should certainly call for serious reflection and caution. Two cases which I investigated recently may illustrate the alteration in bacterial population mentioned above.

The first case involved a 9-month-old infant who appeared initially to be suffering from a respiratory ailment and had a temperature of 104° F. The child was treated with fluids and achromycin. After an early satisfactory response, the child became worse. Chloromycetin and cortisone did not change the course of the disease. Upon the basis of a 1:320 typhi "O" agglutinin titer, it was felt the child was suffering from typhoid fever. Salmonella organisms were never obtained from the child; however, both antemortem and postmortem cultures revealed almost pure cultures of Pseudomonas aeruginosa. Antemortem cultures were from sputum and throat swabs. Postmortem cultures were from lung, spleen, pancreas and intestinal ulcers. The agglutinin titer of the antemortem and postmortem sera against the Pseudomonas was 1:5,120. Here then is an example of systemic infection that could have followed an upset of the host's bacterial population, since this strain of Pseudomonas was susceptible to none of the antibiotics used.

The second case calls to mind not only the possible role of antibiotics in altering bacterial populations but also the changes that may be induced in host susceptibility to certain fungi following cortisone therapy. The effect of cortisone in increasing the susceptibility of animals to experimental fungous infection has been well described by Syverton and his colleagues[13] at the University of Minnesota.

Since the second case, from the beginning, was one of lipoid nephrosis, one could question its use as an example when resistance is to be discussed. However, the fact that cortisone and prophylactic penicillin were used in treatment and the fact that an unusual microbial flora was present at the time of the patient's demise may illustrate a point worth considering.

From postmortem blood, kidney, lung and spleen, very few bacteria were cultured, but an abundance of two fungi was obtained, Candida albicans and

Cryptococcus neoformans. The prognosis in lipoid nephrosis is notoriously poor under normal circumstances, and immunity to cryptococcosis is equivocal at best. One cannot help but raise this question, however: How much did cortisone contribute to upsetting the balance in this case? These remarks are not to be construed as criticism of the handling of this particular case, for the outlook at best was poor. But one cannot help making a mental note of the possible aid cortisone might give to organisms such as these when it is difficult for the host to initiate a satisfactory inflammatory response even under the best of conditions.

Immunity is relative. This second axiom seems as elemental as the first. Yet it represents the hub around which rotate many corollaries, which in turn ramify throughout the subject of immunology and hypersensitivity. These last two terms should not be dissociated, for they are almost always complementary or may work in inverse proportions. The *relative nature* of immunity can be illustrated in the laboratory under controlled conditions. Even an initial immunizing regimen produces some immunity. A more aggressive immunization series may produce a high-level response. We would call patients with a good response solidly immune; yet this immunity can be overridden by a challenge dose if it is big enough.

You see the effects of degrees of immunity in your practice. You do not often see subclinical infections, for their hosts have no complaints; however, we know such infections do occur. The level of immunity in these individuals was adequate; they are discovered accidentally in the laboratory by following the antibody titer rise in the midst of an epidemic of clinical cases. Investigation along this line explains why such a high percentage of adults don't remember having mumps or swear that they did not have mumps—yet they have neutralizing antibodies against the virus. The virus of lymphocytic choriomeningitis, which, because of its name, may be misconsidered as causing neurological infections only, is considered to cause human disease but rarely. Yet this virus, which can cause respiratory illness and systemic infection, must be more common than we think, since 11 per cent of our population were found to have neutralizing antibodies.[1]

The other end of the spectrum of immunity is represented by the feeble response of some individuals to infection. One need not draw only on diabetes, nephrosis and other predicating upsets in physiology. Some individuals continue to be extremely susceptible while our efforts to learn where the hiatus in their immunity makeup lies are fruitless. These are the patients who seem to "pick up everything" and have recurrent infections.

Not enough cognizance is taken of the importance of the antigenic threshold when artificial active immunity is induced. Either too little or too much antigen may be given. If too little is administered, the host fails to recognize the stimulus and builds no antibodies. If too much is given too early, the so-called immunological paralysis of Felton may occur.[3] Repeated adequate doses of antigen cause the host to reach a plateau in the production of antibodies. After maintaining this plateau for a period of time, the titer may actually drop. Antibody production and immunity in general are not static states; they are dynamic and, therefore, relative.

The dose of antigen will vary with its excellence as an antigen. When we consider the small quantity of some antigens given to immunize against certain viral diseases, these also must be considered excellent antigens. Actually, only

a fraction of a microgram may immunize against some of the viral diseases even though the virus is inactivated.

Toxins and other foreign proteins are among the best antigens we have. However, some of the toxins are so potent as lethal weapons that the dose required to immunize is greater than the dose required to kill. This can be shown with tetanus and botulinus toxins in mice. We have been unable to demonstrate antitoxin in humans following their recovery from tetanus even when an attempt was made to show a secondary immune response following a single dose of toxoid. Fortunately, toxoid is available for immunizing against these potent toxins.

The other end of the scale of antigenic excellence is represented by such antigens as insulin. Insulin is a protein derived from a foreign species source, yet it is a very poor antigen. Is it not fortunate that this is so? The incidence of cases of hypersensitivity and intolerance would indeed be great if the reverse were true. Even good antigens in small doses can be masked. A good example of this occurs among the viruses. In the killed virus antigen, the amount of virus is so small in relation to the amount of tissue in which it was grown that the body builds antibodies against the contaminating tissue but not against the virus. This is not true with attenuated live-virus vaccines because the virus grows to reverse the ratio.

It must be remembered that antibody and immunity responses vary with time. It takes time to produce the immune state under natural active circumstances. It is for this reason that the laboratory requires at least two serum specimens spaced a week apart. Sometimes the antibody response requires an even longer time; lymphocytic choriomeningitis is a case in point, requiring three to six weeks for a satisfactory antibody response. Obviously some of these infections are misdiagnosed or not diagnosed because, for one reason or another, later specimens are not collected.

The use of antibiotics may delay or prevent the response of antibodies. Meyer and colleagues[10] demonstrated the tardiness of antibody response in patients with psittacosis who were on antibiotic therapy. Our own experience includes delays up to six weeks. Such comments are not to be misconstrued as a recommendation for delaying the administration of antibiotics for the purpose of obtaining an earlier serologic diagnosis. They do, however, make it clear that serum specimens taken at a later date may be important in viral pneumonitides if an accurate diagnosis is desired. Certainly not all patients with ornithosis will give you a history which includes contact with a parakeet or a parrot, and some will be under your care for six weeks whether you use antibiotics or not.

That the use of effective doses of antibiotics may prevent immunity can be shown experimentally. In our own laboratories, using Diplococcus pneumoniae as the infectious agent and mice as the host, we have been unable to administer penicillin early enough to prevent death and still have a measurable immunity develop. Smadel[12] has shown the relationship between time of administration of antibiotics to the development of immunity in human scrub typhus cases. The incidence of beta hemolytic streptococcus infection, after having dipped to a respectable low with the advent of sulfonamides and antibiotics, is again on the rise. One cannot help but speculate on the general state of human immunity toward acute infections following the introduction of antibiotics.

Again I wish to point out that this is not a crusade against antibiotics, but one has to temper his immediate ideas by long-term views. Immunity levels in the

general population have much to do with the incidence and severity of epidemics. The question could be raised: Are we eventually to boast of being the best treated yet most susceptible group of people in the world? I submit this for your consideration because the introduction of antibiotics-resistant, virulent pathogens, naturally or by subversion with malice aforethought, into a population having a generally low level of natural resistance and/or immunity could prove most serious. Antibiotics are indeed wonderful; however, immunology should not be considered a dead subject.

Unfortunately, not all antibodies are protective. Antibodies producing positive results in test tubes may not necessarily give you an accurate index of the immune state. A patient with histoplasmosis, who was admitted to the University Hospital, comes to mind in this regard. This patient possessed a complement-fixing titer of 1:256, which is high. He was anergic, however, as revealed by the skin test using histoplasmin. The course of his illness was steadily downhill, with a fatal outcome. The patient with systemic Pseudomonas infection, whose case was cited above, died with a 1:5120 agglutinin titer.

It has been our custom to look for antibody in the patient's serum for diagnostic reasons. Actually, in systemic infections the reverse might be more logical. The antigen should be present before the antibody and, if detectable, should permit an earlier diagnosis. Several cases come to mind in this regard but only two will be used. One was that of a psittacosis patient on whom an equivocally positive complement-fixation test was obtained. This same serum specimen used as antigen, in the presence of known positive antibody, functioned satisfactorily at a low titer. This case was followed for six weeks, using weekly serum specimens. The antigen titer of the serum rose steadily over a four-week period before it dropped and the antibody titer rose. One cannot expect to demonstrate antibody if it exists in the presence of excess antigen.

The second case illustrates an unexpected rise in the agglutinin titer of a typhoid case immediately following chloramphenicol therapy. The titer preceding the treatment was 1:80 even though the patient had been ill more than three weeks. Two days following institution of the drug the titer was 1:5,120. The most logical assumption in this instance is that the antibody was almost saturated with antigen. Following therapy, enough of the organisms (antigen) had been removed to permit the assay of antibody that had been there all the time. These situations illustrate that antibody titers are dynamic. It makes considerable difference as to when the specimen is drawn. Antibody levels as well as immunity are relative.

In summary it would seem worth while to repeat these two important axioms for they underlie the proper and intelligent diagnosis, treatment and prognosis of infectious disease, and many hypersensitive states. 1. Microbes exist as populations; therefore the many individuals contained therein offer a great many opportunities for selection and diversified reaction. 2. Immunity is a relative, dynamic state which will vary greatly according to time, inciting agent, accessory factors and serological manifestations. It is unwise to consider either immunity or hypersensitivity with the complete exclusion of the other.

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THE EFFECTS OF INJURY TO THE LATERAL SEMICIRCULAR CANAL

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OUR understanding of the separate functions of the acoustic labyrinth and the relations among its parts had its beginnings in 1824 when Flourens[3] introduced the extirpation method for the study of this structure. Flourens showed that it was possible to injure or remove portions of the labyrinth and thus to eliminate some of its endings while leaving others still in operation. He was able to destroy one semicircular canal and to obtain symptoms due to its absence, but with the continued functioning of the remainder of the system. He noted particularly that hearing was retained after this operation, and it was from this evidence that he made a separation of auditory and equilibratory functions for the inner ear. These experiments were carried out primarily in pigeons, but they were extended by Flourens and his successors to rabbits, guinea pigs, cats, dogs, and other animals. It must be added that the tests of hearing used by these investigators were of the crudest sort, and though they were probably valid in proving the presence of hearing after the operations, they were not serviceable in indicating its degree of normality.

Interest in this field of study has been renewed in recent years because of the development of two types of clinical procedure, (1) techniques for the destruction of the labyrinth as a means of relieving the symptoms of Ménière's disease and (2) the fenestration operation for the treatment of otosclerosis.

A large number of surgical methods have been proposed for the destruction of the labyrinth, as recently reviewed by Cawthorne[2] and by Lindsay and Siedentop.[7] An early method, used by Crockett in 1914, was the extraction of the stapes and the consequent draining of the perilymph. Lempert made the draining more certain by forming two openings, one in the lateral semicircular canal and another in the promontory. Putnam and Day opened one of the semicircular canals, inserted an electrode into the perilymph space, and applied a coagulating current. Lake and several others introduced toxic substances such as alcohol and phenol into the interior of the

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labyrinth. Cawthorne exposed the lateral canal and extracted it with forceps. Goodyear[4] exposed this canal and probed deeply into the vestibule.

The damage to the labyrinth may also be unintentional. Many have reported that occasionally in the course of the fenestration operation an accident has occurred in which the membranous semicircular canal has been injured. Such injuries vary in degree from a barely noticeable tear to the extraction of a portion of the canal.

All of the procedures used for the relief of Ménière's symptoms — draining, coagulating, poisoning, and injuring a canal—nearly always lead to a total loss of both labyrinthine and auditory functions. Most surgeons have reported that the injuries caused accidentally during the fenestration operation have had this ultimate result likewise, though hearing may be present in some degree for a time immediately following the operation. Bateman[1] reported a case of severe damage to the membranous lateral canal as a result of catching it with the dental bur, in which there was a fairly good retention of hearing for at least eighteen days. Cawthorne[2] reported two cases in which a tearing of this canal was followed by a serious impairment of hearing but not its complete loss. Jongkees[5] reported a case in which a portion of the membranous lateral canal was torn away, yet the hearing showed an improvement over the preoperative level after a period of two years.

It is, of course, difficult in these situations to determine in any precise way what the extent of the injury has been. It is even possible to mistake a tearing of the trabeculae of connective tissue in the canal, and their consequent hemorrhage, for an injury to the canal proper. Because of this uncertainty we turn to animal experiments for further information on this problem. Three such experiments have been carried out in recent years.

Schall and Rambo[8] used electrocoagulation after opening the lateral semicircular canal in seven monkey ears. A histologic examination after periods of 10 to 40 days showed widespread destruction of the labyrinth. The organ of Corti was damaged in varying degree in six of these ears, but it appeared normal in one. No hearing tests were given, but the authors concluded from the histologic appearance of the cochlea that any retention of hearing in these animals was improbable.

Schlander[9] carried out similar experiments on three guinea pigs and one rabbit. After the electrocoagulation of a canal these animals soon died, and a histologic examination showed direct effects of the operation in the immediate vicinity of the canal and more general effects in the form of hemorrhages throughout the labyrinth and cochlea. In one animal that survived for a week there were evidences of labyrinthitis also. In this author's view, the losses of general labyrinthine function and of hearing seen in the clinical situation are the result of a slow degeneration initiated by the trauma of opening the canal and inserting an electrode.

Kristensen[6] carried out experiments on guinea pigs, with reflex tests of hearing before and after the membranous lateral canal was exposed and either transected or pulled out with forceps. In some animals the operation was unilateral, and in others it was bilateral. In the animals operated upon unilaterally it was difficult in the tests to exclude hearing in the normal ear, but the attempt was made to plug this ear while observing the reflex movements of the pinna in response to sounds applied to the ear which had been operated on. In general, some hearing was present immediately

after the operation but it gradually became poorer with time. Immediately after bilateral operation, also, the animals usually responded to sounds, and in three instances the hearing seemed practically normal. One animal gave nearly normal responses for eleven days, but after six weeks only traces of hearing remained. No histologic study was made of these ears, but it must be presumed that the operation initiated changes that led eventually to the degeneration of the cochlea.

The present experiments were carried out on seven monkeys. They were anesthetized with sodium pentobarbital injected intravenously, and the lateral semicircular canal was exposed on the right side by the postauricular route. A portion of the membranous canal was then destroyed with a probe or was extracted with a fine hook. In one instance (animal 7) the probe was pushed a short way into the vestibule. In the first five operations, the lumen of the canal was filled with sterile paraffin or bone wax, and paraffin was used also to fill the deep part of the wound. It was found later that this introduction of foreign substances interfered with healing processes, and it was not done in the remaining two operations. Instead, in one of these (animal 6) a plug of cartilage was forced into the canal opening, and in the other (animal 7) the opening was filled with bone dust.

After periods varying from thirteen to sixteen weeks, a second operation was performed on the right ear of five of these animals to permit the recording of cochlear potentials. The round window membrane was exposed, and a platinum foil electrode was placed on it. Another electrode in the form of a needle was inserted into muscle tissue in the facial region. The magnitude of the cochlear potentials was measured with a wave analyzer (a selective voltmeter) on stimulation with tones over the range of 100 to 10,000 cycles. After completion of these measurements, the left ear also was operated upon and cochlear potential tests were carried out in the same manner. The left ears of these animals thus serve as controls. At the end of these tests the animals were perfused through the common carotid artery with Heidenhain-SUSA fixing fluid, after which the right and left temporal bones were removed and prepared histologically by the celloidin method. Also treated histologically were the ears of the two remaining animals that were not tested for cochlear potentials; one of these (no. 2) had suffered a severe middle ear infection, and the other (no. 3) had died of pneumonia.

Cochlear potentials were obtained in all five of the experimental ears in which these tests were made. In one animal (no. 1) they were measured over a range of 700 to 10,000 cycles; in another, (no. 4) from 200 to 10,000 cycles; and in the three remaining animals (nos. 5, 6, 7), over the full range of 100 to 10,000 cycles. The magnitudes of the potentials obtained for particular stimuli varied somewhat for the experimental and control ears. In animal 7 these potentials were consistently smaller for the experimental ear by amounts varying from 6 to 39 decibels, and in animals 1, 4, and 5 they were smaller for this ear by amounts varying from 5 to 15 decibels. In the remaining animal (no. 6) there was no consistent difference between the two ears, but for some tones the right ear was more sensitive and for other tones it was less sensitive.

Figure 1 shows the cochlear potential results for this last animal (no. 6) for both right and left ears. The ordinate scale represents the electrical input to the loudspeaker and not the sound intensity itself. Therefore the shapes of these curves are of no absolute significance,

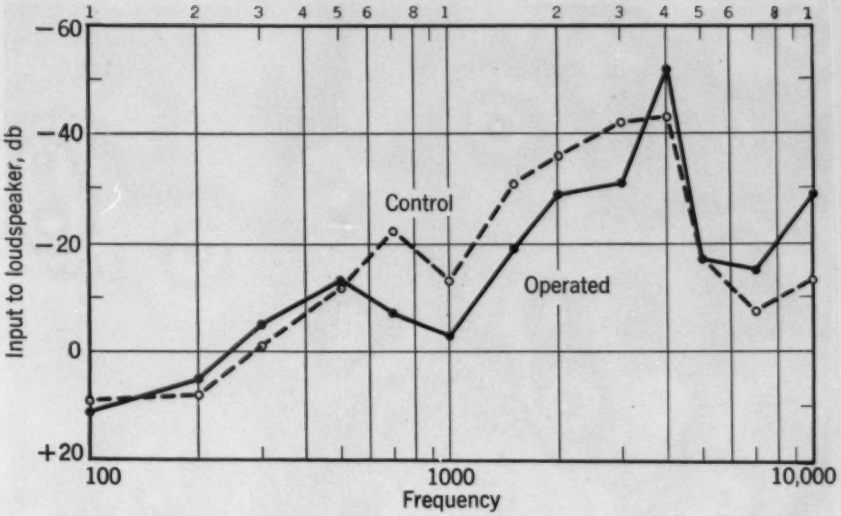

FIG. 1—Cochlear potential curves for both ears of animal 6, showing the relative intensities of stimulation required to produce a response of one microvolt over the frequency range.

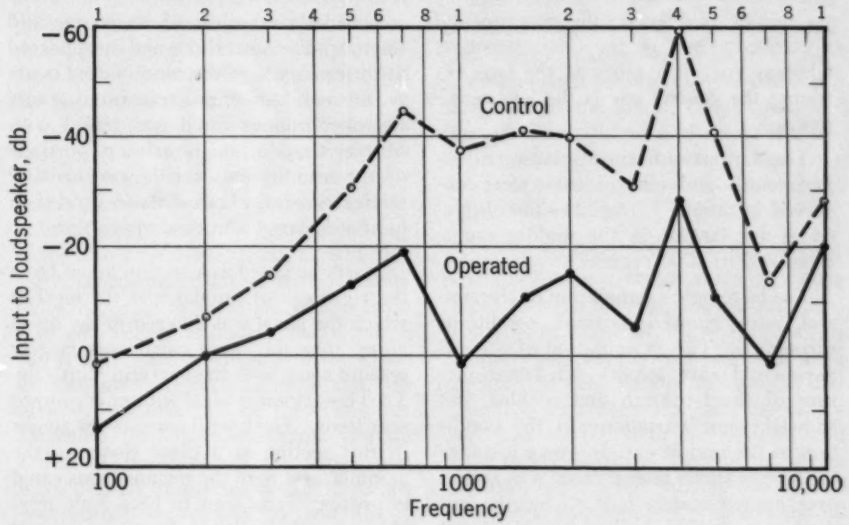

FIG. 2—Cochlear response curves, as in the preceding figure, for animal 7. In this ear the tympanic ring was broken.

FIG. 3—Section from the right ear of animal 6 at the level at which the canal was entered. *la* and *lp* represent two cuts across the lateral canal, *a* one across the anterior vertical canal, and *p* one across the posterior vertical canal.

and only the relations between the two are to be considered. It is clear that for the middle frequencies the experimental (operated) ear is the less sensitive, whereas for most tones at the two extremes the control ear is the less sensitive.

The largest differences between experimental and control ears were observed in animal 7 (fig. 2). The differences are largest in the middle range, from 700 to 4000 cycles.

The histologic examination of the control ears showed normal conditions throughout. The examination of the experimental ears gave a consistent picture of the labyrinth and cochlea, but showed some variations in the condition of the middle ear. In every instance the membranous lateral canal was absent over approximately half its length, and the lumen in this region was obliterated by the growth of scar tissue, which in places was fibrous but more generally was osseous. In general, the scar tissue was dense over most of its course and then rapidly thinned out and disappeared at the ends, where the canal looked nearly normal. At these transition points the membranous canal was sealed with fibrous tissue. The remaining portions of the membranous canals were usually malformed for a little distance, and then assumed almost a normal appearance.

It will be noted in a section taken from the right ear of animal 6 at the level at which the canal was entered in the operation, that the bony canal was largely ground away with the operating bur (fig. 3). This region is filled with unorganized scar tissue. The lateral canal is cut across in this section at a place closer to the ampulla, and here the membranous canal is present. It is seen to be a little misshapen and displaced from its normal position in the bony canal.

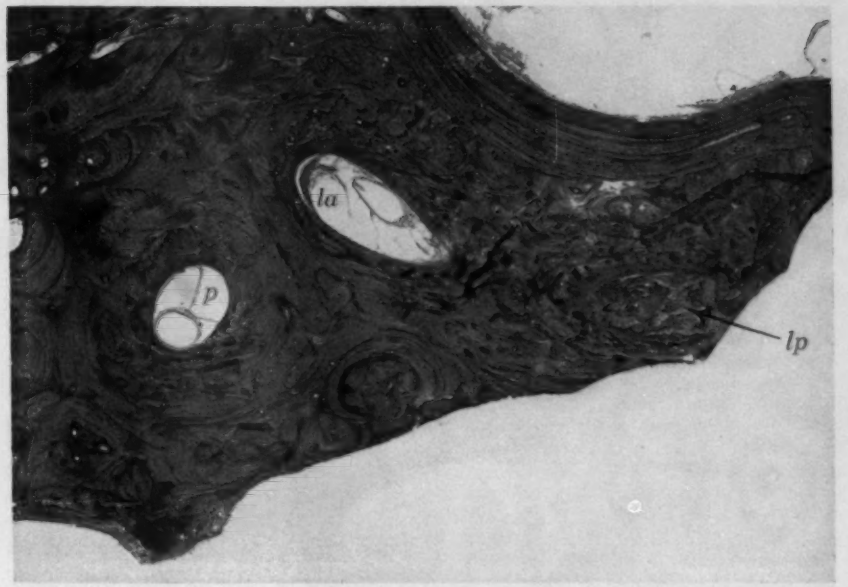

FIG. 4—Section from the right ear of animal 6 at the most lateral position of the lateral canal. The symbols are the same as in the preceding figure.

Figure 4 illustrates a section from the same ear but farther laterally—near the most lateral position of this canal—and at the region of transition from obliterated to patent lumen. Again the canal has been cut across at two places. At one place its lumen is so well filled with bone that its outline can be made out only with difficulty. The other view shows the membranous canal in an abnormal position and surrounded by wisps of adventitious tissue. Close to the ampulla, however, this canal was essentially normal in appearance, and its crista was completely normal. This crista is shown in figure 5 along with the crista of the anterior vertical canal and the utricular macula. All of these endings, as well as the saccular macula and the crista of the posterior vertical canal, were normal in appearance. The cochlea was normal throughout its length (fig. 6).

The conditions represented in these figures were repeated with only slight variations in the other experimental ears, except that in no. 7, in which the probe had been pushed into the vestibule, the crista of the lateral canal was malformed. The adjoining crista of the anterior vertical canal, however, was normal in appearance, as were all the endings elsewhere.

The histologic study provided at least partial explanations for the differences found between the experimental and control ears of some of these animals. In animal 4 the paraffin that had been used to close the deep part of the wound had found its way into the middle ear cavity and had surrounded the ossicles and filled the niche of the round window. Its presence was noted during the second operation, and some of it was removed so that the electrode could be placed on

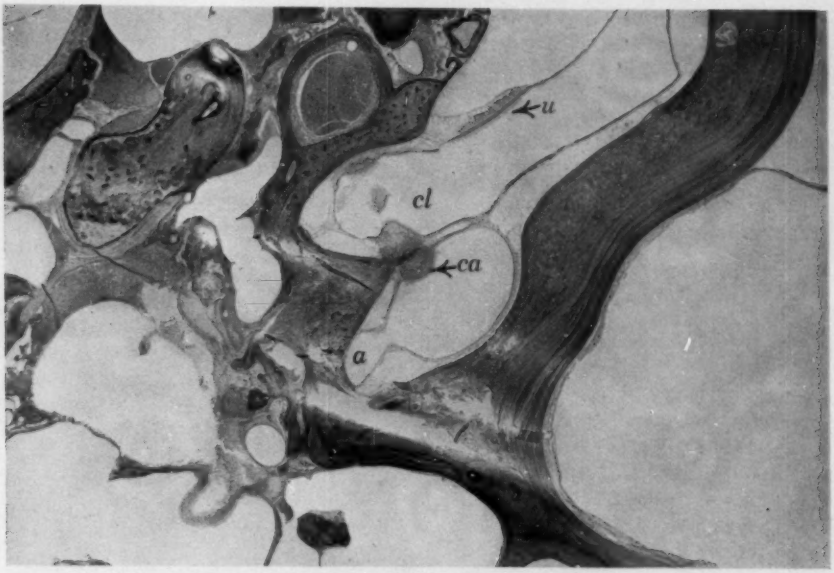

FIG. 5—Section from the right ear of animal 6 showing the ampullae of the lateral and anterior vertical canals with their cristae (*cl* and *ca*), and the macula of the utricle (*u*).

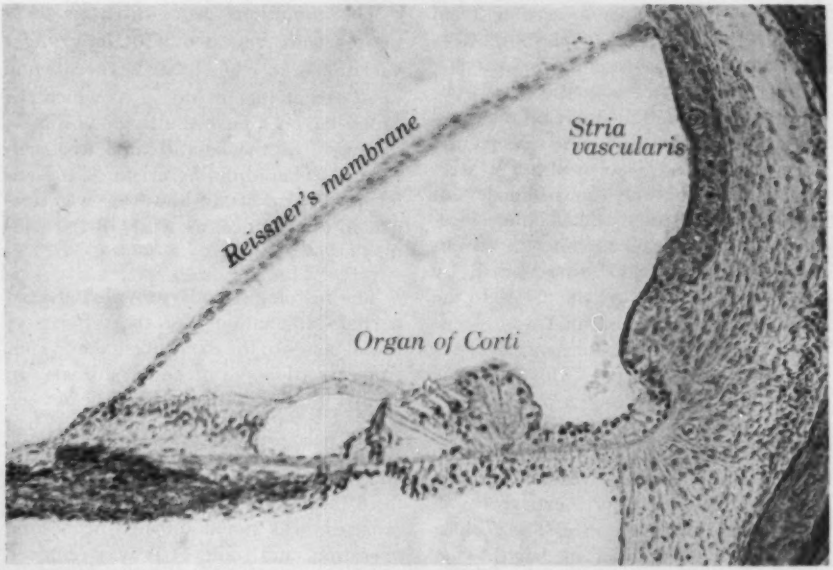

FIG. 6—Section from the right ear of animal 6 showing the sensory structures of the cochlear duct.

the round window membrane. No doubt this material caused considerable restraint of the ossicular movements.

In animals 1 and 7, one edge of the tympanic ring was broken, though with only slight damage to the tympanic membrane. This injury occurred in the approach to the round window. Such injury is difficult to avoid because of the narrowness of the field that the monkey presents here.

In spite of these complications, the cochlear potentials were recorded in the experimental ears over a wide range and at levels that revealed at least a fair performance of the sensory structures. In one of the animals, at least, and possibly in the others, it seems likely that the cochlear action was completely normal. It appears that an injury to a part of the membranous lateral canal in this animal does not necessarily lead to a deterioration of the cochlea, at least for periods up to four months.

These results differ from those observed clinically in man, and the reasons for this difference are not altogether clear. For many of the clinical conditions, like those encountered in the treatment of Ménière's disease, it seems likely that the damage is far more severe than that produced in the monkey experiments. There is greater mechanical injury, and often a more serious loss of perilymph and endolymph. For the lesser injuries we can only speculate at present on possible grounds for differences between monkey and man. One possibility is that the human cochlea is more susceptible in general to damage by the pressure variations and the loss of fluids occasioned by the opening of the membranous labyrinth. Another possibility, which has already been suggested by Cawthorne, is that the human ears may have been in a diseased or enfeebled condition to begin with, and disturbances that normally would have been sustained without damage are now of serious consequence.

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DISCUSSION

FRANZ ALTMANN, M.D., New York: Dr. Wever mentioned that methods could be found to destroy vestibular function in humans without destroying the cochlear function. Arslan in Padua has recorded, I think, 50 or 60 cases in which he did it successfully with ultra short waves, which he directs to the vestibular labyrinth; there was complete loss of the vertigo and preservation of the hearing.

GEORGE E. SHAMBAUGH, JR., Chicago: I have had the experience five times of breaking the endolymphatic labyrinth in doing a fenestration. In each case the tear was easily visible through the microscope. Fortunately, in none of them was there visible bleeding into the labyrinth, and in none of them was the hearing made worse. They all ended up with some improvement, so I am sure that Dr. Wever's observations are applicable to the human, in case the cochlea is fairly healthy.

When we come to Ménière's disease, we have an entirely different situation. A simple fenestration for a patient with Ménière's disease will probably result in a marked drop in hearing. This occurred in an early case which I operated upon by simple fenestration. The patient's hearing practically disappeared along with his attacks of dizziness, which did not recur for about five years. When the dizziness recurred, we did Cawthorne's procedure of extracting the endolymphatic labyrinth. Cawthorne's operation usually results in a dead ear, not only in regard to hearing but to the vestibular function as well. I think the reason for its success in producing a dead ear in cases of Ménière's disease is that we are dealing with an already diseased labyrinth in contrast to otosclerosis, in which there is usually a healthy labyrinth.

FREDERICK HARBERT, M.D., Philadelphia: I should like to ask Dr. Weaver whether or not patency or nonpatency of the ductus reuniens might be a factor.

BENJAMIN H. SHUSTER, M.D., Philadelphia: The presentation of Dr. Wever is based on the premise that the labyrinth is injured during some surgical procedure. It has more implication if one looks at it from a broader clinical point of view—not from the point of view of surgery alone. Many injuries to the labyrinth are not due to surgery. Day in and day out people are exposed to accidents—automobile and industrial accidents—which are followed by a hearing loss. These cases more frequently become problems than those instances of loss caused by surgical procedures.

One is called upon now and then to testify in court in such cases. It is important to determine whether a certain defect in hearing was caused by an accident to the head. I have always felt that as a result of a concussion of the inner ear, there may be rapid degeneration of the hearing, too rapid to be attributed solely to senile or toxic degeneration. I have seen cases in which this was true. I have observed cases of hearing loss in which the patient was re-examined at monthly or bimonthly intervals and showed a further loss of 10 or 15 decibels at each examination; within six or eight months or a year there was almost a complete loss of hearing. This rapid loss suggests to me a traumatic origin, probably due to fibrosis. Dr. Wever's work should explain a good many of the cases of hearing loss which occur following industrial accidents.

Dr. Wever attempts to correlate what happens in experimental animals with what happens in a human being. Those of us who do not work with electrical potentials and other experimental procedures cannot have the confidence in them that the man engaged in research has because we probably do not understand them as well. It seems to me that the indication of a galvanometer in an action potential, as interpreted with reference to a monkey or some other animal, cannot be given as much credence as an audiogram made of the hearing potential of an intelligent human being and based on his subjective report. No doubt the neurophysiologists have as much confidence in their cochlear potentials as I may have in the audiogram, but Dr. Wever did not discuss that point.

KENNETH M. DAY, M.D., Pittsburgh: I cannot refrain from making a few comments, because I personally have operated upon more than a hundred patients with Ménière's disease. I also have had the experience of destroying the horizontal semicircular canal in a few fenestrations.

As for the fenestrations, in none of the cases in which I tore the horizontal semicircular canal was there a complete loss of hearing. Usually the hearing remained near the preoperative level. In one case, the hearing improved to a practical level for speech and has remained at this level for five years. In one case which I operated upon fifteen years ago, the horizontal semicircular canal was accidentally removed by suction. This patient wears a hearing aid in the damaged ear today.

Now, as for the cases of Ménière's disease. My impression has always been that the labyrinth is a tough little organ and is not too easily destroyed. In operations on 51 patients with Ménière's disease, I attempted to destroy the vestibular mechanism but to preserve the cochlea by introducing a fine needle through the horizontal canal into the vestibule and applying an electrocoagulating current. I preserved hearing in 15 cases. The trouble was that when hearing was preserved, the cochlear reaction typical of Ménière's disease usually was preserved also, with hearing distortion

and severe tinnitus. Some of these patients complained so much about the distorted hearing and the roaring tinnitus that I reoperated to destroy the cochlea. Moreover, a few of them still had vertiginous attacks because of incomplete destruction of the vestibular mechanism. I ceased my efforts to preserve hearing for these patients about six years ago and now advocate complete destruction of the labyrinth. To ensure complete destruction, it is necessary to remove part of the membranous labyrinth. I do this by means of a dental broach or fine curet inserted into the vestibule. I strongly urge any of you who attempt to terminate Ménière's disease by labyrinth surgery to be sure that you actually remove part of the vestibular contents. Otherwise you may be surprised because of recurrent attacks of vertigo coming from the posterior semicircular canal and also by the occasional preservation of the hearing disorder.

PHILIP E. MELTZER, M.D., Boston: I must confess that in operations I have done I have not had the good fortune of having hearing retained following injury to the membranous labyrinth during the fenestration operation. As a participant in this experiment with Dr. Wever, I would like to apply these findings in the human, but I can't. It has been my experience that when the membranous labyrinth in humans is injured, there is a loss of hearing. The loss may be sudden or it may be gradual. It finally is worse than it was preoperatively. In these remarks, I am excluding reference to cases involving hemorrhage and secondary labyrinthitis.

It is my belief that the membranous labyrinth is, in many individuals, very sensitive to minimal trauma, so that when it is injured, hearing is likely to become worse than it was preoperatively, even to the point of complete loss in some patients.

EDMUND P. FOWLER, JR., M.D., New York: I think that one of the answers to Dr. Meltzer's and Dr. Wever's remarks is that the increased deafness following fenestration depends on whether a lot of endolymph escapes or not. Recently I had a bad experience with a fenestration of a patient who had considerable dizziness before operation, and again with a patient for mobilization. Both of these cases, I believe, developed severe nerve deafness following operation because of the escape of endolymph. If there is endolymphatic hydrops and the labyrinth is opened, I believe one is much more apt to have a serious loss of hearing following the operation. In the future, therefore, I shall screen my patients more carefully for dizziness before operation.

HAROLD F. SCHUKNECHT, M.D., Detroit: The photomicrographs shown by Dr. Wever reveal that his injuries have produced a remarkable endosteal bone proliferation, the type of reaction which is characteristic of loss of blood supply to the membranous labyrinth. For example, when the internal auditory artery is sectioned, the cochlea, as well as the vestibular labyrinth, fills with connective tissue and bone as a result of proliferation of the endosteum.

It seems reasonable to me that one of the effects of the various labyrinthotomy operations is to compromise the blood supply to a greater or lesser part of the inner ear. As a result of labyrinthine surgery, there might exist in some instances a phlebothrombosis of the cochlear vein or some of its branches. In some instances, a progressive hearing loss can follow injury to the inner ear, and in these cases it may be that a mass of proliferating connective tissue and bone is encroaching on and interfering with the function of vital structures, or that it may be interfering with arterial or venous flow.

Surgical opening of the endolymphatic space does not, in itself, create degenerative changes in the sensory structures. For example, in experiments on animals I have introduced small needles through the round window membrane deeply into the cochlea so as to open the cochlear duct widely in both the first and second turns of the cochlea. Microscopic examination revealed destruction of the sense organ only in the path of the needles, and the audiograms of these animals revealed two dips, corresponding to the two lesions, with normal hearing for other frequencies. Thus, the mixing of endolymph and perilymph does not appear to be catastrophic in itself. I believe that some of the reactions of the labyrinth to trauma may be found in the result of changes in blood supply.

DR. WEVER: Many questions have been raised here which I certainly am not competent to settle. I simply wish to say in reply to Dr. Harbert's suggestion about the patency of the ductus reuniens, that this ductus was patent in our monkey series. In reply to the suggestion of Dr. Schuknecht about the blood supply to the cochlea, I should say also that the blood supply to the cochlea and, indeed, to all of the labyrinth except the lateral canal was not impaired, or at least there was no evidence in our histological studies to indicate any such impairment.

In Memoriam

Truman Leo Boyes.....New York, New York
October 17, 1956

Octavas Dulaney.....Louisville, Kentucky
June 17, 1956

Herbert Graebner.....New York, New York
July 17, 1956

Carl Henning.....Rehoboth Beach, Delaware
October 12, 1956

Henry Kapp.....Haverhill, Massachusetts
July 24, 1956

Samuel N. Key.....Austin, Texas

Charles Royal King.....Toledo, Ohio
June 22, 1956

George Madison Krieger.....Michigan City, Indiana
May 24, 1956

John Sanford Moyer.....Metuchen, New Jersey
July 10, 1956

Kersey C. Riter.....Salt Lake City, Utah
July 22, 1956

George H. Traugh.....Fairmont, West Virginia
September, 1956

TRANSACTIONS

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American Academy of Ophthalmology and Otolaryngology,
100 First Avenue Building, Rochester, Minnesota

HIGHLIGHTS OF THE 1956 CONVENTION

The Sixty-First Annual Meeting of the Academy was the occasion for the announcement of an important step in the prevention of blindness. Glaucoma has been placed in the category of progressive chronic diseases by the Public Health Service, and the chief of the Chronic Disease Program in the Division of Special Health Services, Dr. Arnold B. Kurlander, told of the plans now under consideration for the screening of millions of people through Public Health programs. In this way they hope to narrow the field for a practical endeavor by ophthalmologists to examine critically the doubtful and probable cases of early glaucoma. "The impact of chronic disease on our population is now recognized as the nation's No. 1 public health problem. Glaucoma, estimated to strike 2 per cent of all people over 40, is a prime example of a chronic progressive disease. A preliminary protocol has been drafted and is now under study by our consultants. We hope to perfect arrangements with leading eye clinics so as to initiate cooperative studies in areas identified as significant by the consultant." With early detection of glaucoma, we may confidently hope to save from blindness many more victims of the malady that heretofore has been possible.

A comprehensive discussion of postoperative cataract complications covering pathogenesis, clinical manifestations, pathology and treatment by Drs. Michael J. Hogan, John M. McLean and A. E. Maumenee will be augmented and published as a manual by the Academy or expanded into a text. This symposium was the culmination of three years of work by the authors and was condensed for presentation at a scientific session. In this work will be found answers to many perplexing problems encountered by all ophthalmic surgeons.

At the present time *allergy* permeates the field of the otolaryngologist with a greater degree of saturation than it does that of the ophthalmologist, though it is universal and no medical program can be constructed without an Allergy plank or a whole platform of soundings, findings, and clinical applications. The symposium on "Allergy of the Ear," by Drs. Herbert Rinkel, Edley Jones, Eugene L. Derlacki, and Raymond E. Jordan, was another Academy presentation by a select group of artists who made an orderly display of proof in a field of uncertain disease complications that have both systemic and local manifestations. The papers will be published in full in the TRANSACTIONS.

A number of valuable single papers were presented, new instruments described and motion pictures shown that carried the scientific program to new heights.

The scientific exhibit was acclaimed as the best in history of the Academy. A trio of experienced judges made the awards on the usual basis. In ophthalmology, first award went to Dr. David O. Harrington for his elaborate demonstration in "Visual Field Examination"; second award went to Drs. Algernon B. Reese and Bradley R. Straatsma for a colorful display of gross and microscopic pictures of "Precancerous and Cancerous Melanomas"; third award went to Drs. Peter Kronfeld and H. Isabelle McGarry for a presentation of cases showing "The Effect of Glaucoma Operation upon Aqueous Dynamics."

In otolaryngology, first award went to Drs. Irving M. Blatt, Philip Rubin, James H. Maxwell and John E. Magielski for an interesting display on "Secretory Sialography in Health and Disease"; second award went to Dr. William T. K. Bryan and Marian P. Bryan for a kodachrome study of "Cellular Pathology in Diseases of the Ear, Nose and Throat"; third award went to Drs. Max L. Som and Leon M. Arnold, whose exhibit included colored illustrations of "Hemilaryngectomy: A Technique for Cordal Carcinoma with Extension Posteriorly." Many other excellent exhibits and demonstrations attracted the attention of hundreds of interested students and doctors.

The substitution of the Annual Dinner for the Evening of Diversion on Monday evening met with encouraging approval, and the Program Committee has read the desires of the members in the attendance at the one planned night of entertainment. An excellent dinner followed by an unusual performance by Paul Hartman with Betty Luster and Undine Forrest, Alcetty, and the Four Encores put the event on record as one of the best. Our thanks are extended to Ralph Ginsburgh and the Palmer House staff for making the banquet an attractive picture in the album of Academy conventions.

The highest award of the Academy given to instructors and contributors to the scientific program, the scientific exhibits, and the Home Study Courses is the Award of Honor, earned on points scored by a system adopted by the Council four years ago. Honor awards were presented to the following Fellows of the Academy:

Franz Altmann
Moacyr E. Alvaro
Karl W. Ascher
Raynold N. Berke
Frank D. Costenbader
Isadore Givner
Dewey Katz
Brendan D. Leahey
Alden H. Miller
Lewis F. Morrison
Ralph Harrison Pino
Aubrey G. Rawlins
Hunter H. Romaine
George V. Simpson

A resolution to change the Constitution and By-Laws of the Academy in regard to classification of membership was given its first reading and will be voted upon at the next meeting. A new category of an associate membership will make it possible for many teachers in the basic sciences who are not medical graduates to become more closely affiliated with the Academy and its educational program. Such associate members shall pay dues equal to one half of the junior membership dues and shall receive the TRANSACTIONS, but they shall not vote or be eligible for office.

American Academy of Ophthalmology and Otolaryngology
CONVENTION ATTENDANCE — OCTOBER 14-19, 1956

STATE	MEMBERSHIP AS OF 4-1-56	FELLOWS	GUESTS	RESIDENTS	CANDIDATES	ORTH. TECH.	TOTAL
Alabama	61	27	7		2	1	37
Arizona	30	7	2				9
Arkansas	25	12	6		1	1	20
California	619	156	24	5	14	5	204
Colorado	86	37	8	1	1	3	50
Connecticut	104	31	4	1	1	1	38
Delaware	14	5	1				6
Dist. of Columbia	87	24	10	3	1	5	43
Florida	146	46	10	2	1	2	61
Georgia	72	34	11		2	2	49
Idaho	17	6	1		1		8
Illinois	359	287	218	100	9	8	622
Indiana	133	94	38	7	4	3	146
Iowa	83	48	32	8	2	2	92
Kansas	60	28	8	3	1		40
Kentucky	63	40	15	1	1		57
Louisiana	78	42	16	3			61
Maine	37	6	1			1	8
Maryland	81	40	12	9		4	65
Massachusetts	208	61	13	1	1	1	77
Michigan	228	142	55	23	4	12	236
Minnesota	136	63	19	6	5	3	96
Mississippi	40	13	4		1	1	19
Missouri	166	73	31	7	2	4	117
Montana	18	8	6		1		15
Nebraska	47	26	3	1	1	1	32
Nevada	8	1	1		1		3
New Hampshire	18	7	2				9
New Jersey	199	70	19	2	2		93
New Mexico	15	3					3
New York	913	278	60	16	5	12	371
North Carolina	82	28	2	1	1	1	33
North Dakota	17	7	3				10
Ohio	338	207	58	13	5	8	291
Oklahoma	40	20	5	1	1	1	28
Oregon	66	14	2			1	17
Pennsylvania	480	177	41	10	6	4	238
Rhode Island	22	6					6
South Carolina	38	17	7		1		25
South Dakota	15	11	5				16
Tennessee	98	46	11	2	2	3	64
Texas	253	105	17	4	7	1	134
Utah	32	10	3				13
Vermont	12	3		4			7
Virginia	92	26	5		2	1	34
Washington	84	20	9	3		1	33
West Virginia	48	24	1		1		26
Wisconsin	106	82	30	8	3	6	129
Wyoming	10	5	1				6
U. S. Territories	38	12	6				18
Canada	151	62	32	4	10	8	116
Foreign	41	6	43	3	1		53
TOTALS	6214	2603	918	252	104	107	3984
Ladies							1099
Technical Exhibitors							648
GRAND TOTAL							5731

TEMPORAL BONES FOR MICROSCOPIC STUDY

I. Technique for Removing Temporal Bones

Modified from description by Stacy R. Guild, Ph.D., Johns Hopkins University, as published in Nelson Loose-Leaf Medicine of the Ear, page 663. Edited by Edmund Prince Fowler, Jr., M.D., New York.

IT is easier to remove the portion of the temporal bone needed for histologic study of the inner and middle ear than it is to describe the technique of removal. The following account, however, contains the essential points:

1. In all procedures, care must be exercised to remove the bone in a way that will not permit collapse of the head, unless it is definitely known at the time of necropsy that the body will not be viewed later by relatives or friends of the deceased.

2. All major cuts should be made with a saw instead of with a chisel, because extensive fracturing of the bone is caused by the wedge-shaped chisel.

3. All necessary cuts can be made with a "rocker" saw, of the type usually provided in autopsy rooms, made by the Orthopedic Frame Company, Kalamazoo, Michigan. The cuts may also be made with a motor-driven circular saw of suitable size. Saw blades for this purpose should not exceed 2⅜ inches in diameter; otherwise there usually will be difficulty in getting the saw into the posterior cranial fossa deeply enough to make cut 4 (fig. 1) at the proper level. (Even a one-inch saw blade can be used.) The nut and washer assembly used to fasten the saw blade to the revolving shaft must be small enough to permit a deep cut to be made. The saw can be driven either by a flexible shaft or directly by the shaft of a small motor.

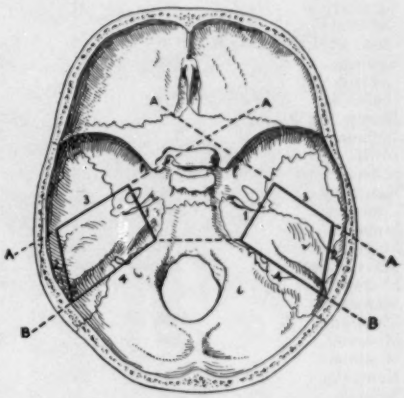

FIG. 1—Solid lines indicate essential saw cuts 1, 2, 3, and 4, described in the text. Dotted lines show saw cuts which can be made if portions of the bone and eustachian tube are removed with a straight hand saw. The latter method requires that the skull be filled with plaster of Paris after removal of the temporal bones.

4. After the skull cap and the brain have been removed, the parts of the inner and middle ear needed for histologic studies can be removed intact by proceeding thus:

(a) Four cuts are made with a saw. Cut 1 and cut 2 are made at right angles to the superior angle of the petrous pyramid. Cut 1 is made as near the apex of the petrous bone as the size and shape of the posterior cranial fossa permit; in any case it is well anteromedial to the cochlea. The farther "forward" this cut is made, the larger is the portion of the eustachian tube obtained for study. If the entire eustachian tube is desired, this cut can be eliminated altogether; cuts are then made vertically in the sphenoid bone in front of the sella turcica and through the body as it connects with the occipital bone. Cut 2 is

made roughly parallel to cut 1 and through the middle of the mastoid region as near to its lateral wall as the saw can be held. (For most histologic studies it is not necessary to obtain all of the mastoid process.) Cut 3 is made "vertically" in the floor of the middle cranial fossa about one inch in front of the petrous ridge and laterally as close to the cranial wall as the saw will go. This cut should join the forward ends of cuts 1 and 2. If properly made, cut 3 is about 3/8 inch lateral and in front of the tympanic membrane, and the block of tissue removed includes the deeper part of the osseous external canal. Cut 4 is an "undercut" of the block of tissue outlined by the preceding three cuts. It is made as nearly as possible in the anatomically horizontal plane and as near to the floor of the posterior cranial fossa as the saw can be placed; usually it is possible to make the cut pass through or below the dural exit of the vagus nerve. The posterolateral end of cut 4 should be made first; thus it is possible to carry the cut nearer to the apex of the petrous portion of the bone than is the case when the cut is started at the narrow anterior part of the posterior cranial fossa.

(b) Removal of the block outlined by the four saw cuts is often the most difficult part of the entire procedure. If the cuts have not overlapped at the "deep" corners (and often they do not), the block of tissue remains held in situ not only by some bony connections but also by the dense fibrous tissue of and near the medial side of the mandibular articulation. Attempts to remove the block forcibly by the use of ordinary large forceps or by prying with a wedge usually result in crushing pneumatized areas or in making a crack along the "plane" of the middle ear. The best instrument for use in removal of the outlined block is a "lion-jawed" forceps which has been modified by making a deep "central" notch in each jaw and thinning each of the four resultant corners. The block is grasped by placing the two prongs of one jaw of the forceps in cut 4 and closing down the other jaw in such a manner that its prongs grasp the superior surface of the block just lateral to the superior angle of the petrous pyramid. This region of the superior surface is usually more free of underlying pneumatized or marrow spaces than are the more lateral portions of the superior surface. With the block grasped as described above, gentle back-and-forth movement of the handles of the forceps causes the unsevered bony connections at the "deep" corners to break without damage to important structures. After the bony connections are broken, while continuing to grasp the block with the forceps, it can be twisted enough to permit insertion of a knife along cuts 3 and 4 and severance of the dense fibrous connections remaining. A wide sharp chisel will often prove helpful, especially in severing the styloid process.

With proper tools and a bit of experience, the surgeon can remove both (right and left) temporal bone blocks in good condition in about twenty minutes after the brain has been removed.

The carotid arteries should be tied to facilitate injection by the undertaker. This may be done either by drawing the carotid up into the base of the skull, then ligating the vessel and allowing it to retract into the neck before the bone is removed, or by ligating the cut ends of the arteries after the bone is removed.

II. Technique of Fixation

Formalin Solution

Immediately after the block of bone has been removed, it should be placed into 400 cc. of 20 per cent formalin (one part commercial formalin to four parts water). In diluting the formalin it is preferable to use water which has been degassed in vacuum, because it will absorb air bubbles trapped in the middle ear and mastoid and will facilitate penetration of the fixing fluid.

Second and third changes in 10 per cent formalin should be made on the following two days. The blocks can be kept indefinitely in formalin. The best container for the bones is a wide-mouthed one-pint Ball fruit jar.

In cases in which the histopathologic changes in the inner ear are likely to be of primary importance, more rapid fixation can be obtained if the stapes is removed. This may be undesirable in cases in which the pathologic tissue changes about the stapes footplate are of primary importance.

III. Instructions for Shipping and Method of Handling Temporal Bones

As suggested by the Subcommittee on Research of the Committee on the Conservation of Hearing of the American Academy of Ophthalmology and Otolaryngology.

After the bones have been in 10 per cent formalin solution for two or more days, they may be prepared for shipping. They may be packed in an airtight fruit jar by filling the intervening space with absorbent paper and pouring off the excess fluid. The absorbent paper holds enough fixative to keep the bones moist.

The office of the Executive Secretary-Treasurer of the Academy will supply on request full information regarding shipping and a list of the laboratories which are prepared to process the bones.

Information to Accompany the Bones

It is suggested that a complete identification and history of the case be furnished along with the temporal bones, including the diagnosis of the terminal illness, a copy of the autopsy report, the time of death and the time of removal of the specimens. The otologic history, together with the records of functional tests, is of particular importance. This history can be filed with the tissue sections.

Method of Handling the Sections

A list of the laboratories which are prepared to section temporal bones will be furnished upon request.

The procedure suggested by the Committee is that the bones will be sectioned serially and every fifth section mounted and stained. These stained sections will be divided into three sets. Each set will then contain every fifteenth section.

The otologist who furnishes the bones will receive one set of slides. The second, "tracer," set will be retained in the laboratory, and the third set will be reserved for collection in some central place where all such specimens will be available for study. The remaining unmounted sections will be retained in the laboratory, to be available as needed for special stains, et cetera.

The laboratory and the otologist concerned will have the privilege of making a preliminary report on the histopathologic findings. In addition, the sections will be available for complete study and review along with other cases at some later time.

JOHN R. LINDSAY

INTERNATIONAL ASSOCIATION OF SECRETARIES OF OPHTHALMOLOGICAL AND OTOLARYNGOLOGICAL SOCIETIES

A Message from Japan

The biggest difficulty for Japanese physicians in relation to attendance at medical congresses is the disadvantage we have because of the current exchange rate. Without the help of the government, some philanthropic organization, a pharmaceutical company, or some other assistance we cannot possibly hope to attend a congress held overseas. Very few Japanese are wealthy enough to be able to afford the expense for a trip across the water.

Therefore, our ardent wish is for a congress to be held in Japan, or somewhere in the Orient.

Even then, however, it will not be without considerable difficulties, particularly because of the large expense involved in such an undertaking. The raising of this money would be an exceedingly heavy burden for the medical circle in Japan. And so I have been wondering if it might not be possible to hold a congress without some of the entertaining.

According to our experience, most of the expense incurred is in social diversions for foreign scholars. If, however, we could abolish the custom of entertaining our foreign guests and spend the money solely for better equipment of the place where the meeting is to take place, I believe that we too can hold a congress in our country. And I hope my wish will come true in the near future.

As a matter of fact, there is a time-honored way of thinking that we in Japan should do all we can to entertain foreign scholars who visit our country. Since, however, there exists this great economic hardship attributable to the past war, it would be very difficult for us to live up to this custom.

Japan is, as you know, a small island country apart from other nations. Not only because of that but because of the language barrier, the research work of Japanese scholars has frequently little opportunity to become known in foreign countries. And it is deplorable when I think that important research is being conducted in Japan, for us alone. This work should be communicated to medical circles throughout the world.

What we should like to know is whether or not foreign scholars would be willing to attend a congress, if held in Japan, even though material service and entertainment is not offered in profusion.

On this occasion I should like to mention that there are some unique sights to be seen in Japan. I believe Japan can be a suitable place as a first leg on a tour through the Far East.

As to Japanese scholars, we are always ready to become friends with our foreign counterparts despite the economic difficulty and the language barrier. It would give us great pleasure to be of service as your hosts.

TOSHIKAZU NISHIHATA, M.D.

NOTICE

Academy Fellows who might wish to attend a congress in Japan will please write to Paul C. Craig, M.D., 232 North Fifth Street, Reading, Pennsylvania.

IN MEMORIAM

October 1955 — October 1956

Charles Joseph Adams, Kokomo, Ind.
Thomas Dyer Allen, Evanston, Ill.
R. Winfield Baeseman, Allenhurst, N. J.
Irving Francis Barnett, Chicago, Ill.
Louis J. Beyer, Buffalo, N. Y.
Egil Boeckmann, St. Paul, Minn.
James Bordley, Jr., Baltimore, Md.
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Arnold Herman Knapp, New York, N. Y.
Adolph Krebs, La Mesa, Calif.
Robert Scott Lamb, Washington, D. C.
James William Lehan, Greeley, Colo.
Harry Sumner Lester, Streator, Ill.
Bert G. Levin, St. Paul, Minn.
Frank E. Mallon, Brooklyn, N. Y.
John McGuire, Bluefield, W. Va.
Tom O. W. Meissner, Athens, Ga.
Samuel Archard Morris, Los Angeles, Calif.
Wilfrid Edward Muldoon, San Antonio, Texas
George Waverly Nairn, Warsaw, N. Y.
Charles Francis Nutter, Nashua, N. H.
John Nelson Osburn, Corona del Mar, Calif.
Robert Wright Owsley, Arcadia, Calif.
James Devote Perdue, Mobile, Ala.
James Ross Reed, Pasadena, Calif.
Clarence Edward Robbins, Pierre, S. D.
William Humes Roberts, La Jolla, Calif.
Henry Paul Schugt, New York, N. Y.
James Washington Smith, New York, N. Y.
William Hamilton Smith, Hagerstown, Md.
Carl Brosius Sputh, Indianapolis, Ind.
Herman Marshall Taylor, Jacksonville, Fla.
Peter Hunter Thompson, Boston, Mass.
Robert Russell Tracht, St. Paul, Minn.
Ivan Ford Weidlein, Cleveland, Ohio
Samuel Brownell Westlake, St. Louis, Mo.
Anthony Charles Zehnder, Newark, N. J.

Necrology

CHARLES JOSEPH ADAMS

Dr. Charles J. Adams, retired eye, ear, nose and throat specialist, died April 7, 1956, at the age of 73 years at Kokomo, Indiana. He had been in ill health since October 1949.

Dr. Adams was born at Lafayette, Indiana, on June 3, 1883. On October 5, 1913 he was married to Lovie Hobbs, who survives.

In his early childhood, Dr. Adams' parents moved to Warren, Indiana. After his graduation from Warren High School he entered the medical school of Purdue University, which later became affiliated with Indiana University School of Medicine, and received his degree as Doctor of Medicine in 1906. He then entered the special field of ophthalmology and otolaryngology, taking postgraduate work at City Hospital and the Methodist Hospital in Indianapolis and in 1907 and 1908 at the Chicago Eye, Ear, Nose and Throat Hospital. In 1923 and 1924 he spent a year in advanced study at Harvard University, Boston, at the Royal London Ophthalmic Hospital in London, England, and at the Medical University of Vienna, Austria.

Dr. Adams was a very active civic leader and did much to stimulate a program of medical aid for underprivileged children. In later years he became deeply interested in the work of the Rotary Foundation, which provides international scholarships for outstanding young men and women.

A member of the American Academy of Ophthalmology and Otolaryngology, Dr. Adams also held membership in the American and Indiana State medical associations and Howard County Medical Society. He was an emeritus member of the Chicago Laryngological and Otological Society, and was a diplomate of the American Board of Otolaryngology.

Dr. Adams was devoted to the medical profession which he served with conspicuous loyalty and whose best standards he upheld zealously. He will be remembered as a kind gentleman and a warm-hearted friend.

THOMAS DYER ALLEN

Dr. Thomas D. Allen died February 14, 1956 at Evanston Hospital, Evanston, Illinois. He was 67 years of age.

Dr. Allen was born April 18, 1888, at Omaha, Nebraska. He attended the University of Wisconsin in 1913 and received his Medical Degree from Rush Medical College, Chicago, in 1915. He then became associated with Dr. W. H. Wilder of Chicago. Postgraduate work was done in Vienna, Austria, in 1926.

Dr. Allen was emeritus clinical associate professor of ophthalmology at Rush Medical College, University of Illinois College of Medicine. He was attending ophthalmologist at Cook County Hospital and at the Illinois Eye and Ear Infirmary in Chicago, associate ophthalmologist at the Presbyterian Hospital in Chicago, and was on the courtesy staff of Evanston Hospital.

Dr. Allen was a specialist certified by the American Board of Ophthalmology, past president of the Chicago Ophthalmological Society and a Senior Fellow of the American Academy of Ophthalmology and Otolaryngology, and had served on its Board of Councillors from 1947 to 1950. He was also a member of the American Ophthalmological Society, the Chicago Pathological Society, Sigma Xi, and the Association for Research in Ophthalmology; a fellow of the American College of Surgeons, and of the Institute of Medicine of Chicago; secretary-treasurer for North of Panama of the Pan American Association of Ophthalmology; a member of the American Medical Association, Chicago Medical Society, and the Illinois State Medical Society. He served in France during World War I.

Dr. Allen's contributions and services in ophthalmology and otolaryngology will be remembered with great admiration by all of his associates.

Survivors are his wife, Ruth Delzell Allen, six children and thirteen grandchildren.

R. WINFIELD BAESEMAN

The death of Dr. R. Winfield Baeseman on February 1, 1956, in Bandah, India, is without question a great loss to the field of ophthalmology. He died at the age of 59 years from a heart attack while on his tenth tour of duty as an eye surgeon at the Church of Scotland Mission. It was his custom to furnish quantities of surgical supplies, sutures, and instruments for each trip, and he left them for the benefit of the Mission Clinic. He gave financial help to promising college students in India. He operated upon hundreds of natives for cataract extraction.

Dr. Baeseman was born in Wausau, Wisconsin. He graduated from Marquette University and received his medical degree from the University of Louisville School of Medicine, and his degree of Master of Science in Ophthalmology from the University of Pennsylvania Graduate School. He first practiced his specialty as an associate of Dr. James A. Fisher, Sr., in Asbury Park in 1931. Five years later he established his own practice in Asbury Park, and later moved to Allenhurst, New Jersey. He was licensed to practice ophthalmology in New Jersey, New York, Florida, Pennsylvania and Indiana.

Dr. Baeseman interned at the Indianapolis General Hospital; he was ophthalmologist at the Fitkin Memorial Hospital, Neptune, N. J. and at the Marlboro State Hospital in Marlboro, N. J., and was consulting ophthalmologist to several other institutions. He was a member of many medical societies, including the Monmouth County Medical Society, the Medical Society of New Jersey, the American Medical Association, the American Academy of Ophthalmology and Otolaryngology, the American College of Surgeons, the International College of Surgeons, the Pan American Association of Ophthalmology, and the New Jersey, New York and Pennsylvania Association of Ophthalmology.

Dr. Baeseman served in World War I, and was a 32nd Degree Mason. He is survived by two sisters and several nieces and nephews.

IRVING FRANCIS BARNETT

Dr. Irving Francis Barnett was born in Chicago, April 29, 1892, and died in that city on June 26, 1956.

After preliminary education at Northwestern Academy, Dr. Barnett entered the University of Illinois College of Medicine, Chicago, which granted him the degree of Doctor of Medicine in 1913. He established his practice in Chicago and soon decided to specialize in otolaryngology, restricting his practice to that branch of medicine in 1917. Later, he included the practice of ophthalmology, and he eventually became a diplomate of both the American Board of Otolaryngology and the American Board of Ophthalmology.

In Chicago, Dr. Barnett became attending surgeon at the Illinois Eye and Ear Infirmary and attending otolaryngologist at Chicago Polyclinics Hospital and at Henrotin Hospital. Later in his career, Dr. Barnett became senior ophthalmologist at Henrotin Hospital and associate clinical professor of ophthalmology at Stritch School of Medicine of Loyola University. For two years he was county jail physician. During World War I, he served with the British Army. For his medical service, he was awarded the Victoria Cross.

Dr. Barnett was elected a Fellow of the American Academy of Ophthalmology and Otolaryngology in 1933. He was a member of the American Medical Association and of the Association for Research in Ophthalmology, and a fellow of the American College of Surgeons. He was a member of Our Lady of Mt. Carmel Catholic Church.

Surviving Dr. Barnett are his wife, Harriet, and their son, John Ayres.

LOUIS J. BEYER

Dr. Louis J. Beyer was born in Buffalo, New York, on June 1, 1876, and died in that city on April 8, 1956.

The University of Buffalo granted Dr. Beyer the degree of Doctor of Medicine in 1899. He subsequently served for many years on the faculty of his alma mater. He also served as chairman of the city board of health and as a member of the staffs of Millard Fillmore, Edward J. Meyer Memorial, and Children's hospitals.

Dr. Beyer was elected a Fellow of the American Academy of Ophthalmology and Otolaryngology in 1918, and attained the status of Life Fellow in 1948. He was an associate member of the American Medical Association, a fellow of

the American College of Surgeons and a diplomate of the American Board of Otolaryngology.

EGIL BOECKMANN

Dr. Egil Boeckmann, of St. Paul, Minnesota, died in Dellwood, Minnesota, on September 3, 1956, at the age of 73.

A graduate of the University of Pensylvania Department of Medicine, Philadelphia, Dr. Boeckmann received his medical degree in 1905 and was licensed to practice in 1906.

Certified as a specialist by both the American Board of Ophthalmology and the American Board of Otolaryngology, Dr. Boeckmann was elected to the American Academy of Ophthalmology and Otolaryngology in 1918. He became a Life Fellow of the Academy in 1948. He was also a member of the American Medical Association.

JAMES BORDLEY, JR.

Dr. James Bordley, Jr., was born February 20, 1874, at Centreville, Maryland. He was graduated from Davis Military Academy, and received his degree in medicine in 1896 at the University of Maryland. Postgraduate work was done in Vienna in 1903.

Dr. Bordley was on the staff of the South Baltimore Eye, Nose and Throat Hospital, Hospital for the Women of Maryland, the Union Memorial Hospital, and the Church Home and Infirmary.

Dr. Bordley was a Life Fellow of the American Academy of Ophthalmology and Otolaryngology, a member of the American Ophthalmological Society, and past-chairman of the American Medical Association (Ophthalmological Section), as well as a member of the Episcopalian Church. He served in World War I with the rank of Lieutenant Colonel.

Besides being extremely interested in Colonial history, Dr. Bordley spent much time learning about American antiques, and became an ardent collector.

Dr. Bordley died January 7, 1956, in Baltimore, Maryland. Survivors are his wife, Margaret C. Bordley; two sons, Dr. James Bordley, III, of Cooperstown, New York, and Dr. John E. Bordley of Baltimore, who is also a Fellow of the American Academy of Ophthalmology and Otolaryngology; one daughter, Mrs. Charles A. Webb of Baltimore; and seven grandchildren.

EDGAR OSGOOD BREAKSTONE

On November 3, 1955, Dr. Edgar O. Breakstone died at Michael Reese Hospital, Chicago, at the age of 47 years.

Dr. Breakstone graduated from the University of Pittsburgh in 1930 and received his medical degree from the Chicago Medical School in 1935. Postgraduate work was done at the University of Illinois. He was a resident physician at Cook County Hospital and from 1944 to 1949 was on the faculty at the University of Illinois Medical School, later joining the faculty of the Chicago Eye, Ear, Nose and Throat Hospital.

Dr. Breakstone was a diplomate of the American Board of Otolaryngology, a Senior Fellow of the American Academy of Ophthalmology and Otolaryngology, and a member of the Pan American Association of Ophthalmology and of the American College of Allergy.

His wife, Alta, survives with one son, Jeffrey, and one daughter, Jill.

JOHN MACKENZIE BROWN
1878-1955

(See Eulogy in the TRANSACTIONS, January-February, 1956, pp. 154-156.)

HANS BRUNNER
1893-1955

(See Eulogy in the TRANSACTIONS, March-April 1956, pp. 220-221.)

JAMES LESLIE BRYAN

Dr. James L. Bryan was born December 14, 1886, at New Middleton, Tennessee. His preliminary education was obtained at Bethel College, Russellville, Kentucky. He received his medical degree at Vanderbilt University School of Medicine, Nashville, in 1910. He began the practice of medicine in Nashville in 1912, and later went to New York, where he spent four years in postgraduate work in otolaryngology.

A Senior Fellow of the American Academy of Ophthalmology and Otolaryngology, Dr. Bryan was also a member of the International College of Surgeons, the Nashville Academy of Medicine, and the Nashville Academy of Ophthalmology and Otolaryngology. He was a diplomate of the American Board of Otolaryngology. He was on the staffs of Vanderbilt University Hospital, St.

Thomas Hospital, Mid-State Baptist Hospital, and Nashville General Hospital, all in Nashville.

Dr. Bryan was a member of the Baptist Church and was interested in the work of the Baptist Orphanage. He served his country during World War I.

On December 20, 1955, Dr. Bryan died of coronary thrombosis. His wife, Neine Bullington Bryan, survives.

MIRIAM M. BUTT

Dr. Miriam M. Butt was born August 14, 1872. She attended Friends Central School, Philadelphia, and received her degree in medicine at the Woman's Medical College of Pennsylvania in 1894. She began private practice in Philadelphia.

Dr. Butt was a Life Fellow of the American Academy of Ophthalmology and Otolaryngology, and a member of the American Medical Association and of state and county medical societies. She had been on the staff of the Woman's Hospital, Philadelphia.

Dr. Butt died May 19, 1955, aged 82 years.

LUCIAN ORVILLE CLEMENT

Dr. Lucian O. Clement was born September 28, 1875, at Waseca, Minnesota. He graduated from the University of Minnesota in 1899 with a Bachelor of Science degree, and in 1902 received his degree as Doctor of Medicine. In 1902 he began private practice in Lamberton, Minnesota. Later he did postgraduate work in New York, Boston, Vienna, and London.

Dr. Clement was on the staffs of the Salem General Hospital and the Salem Memorial Hospital, Salem, Oregon. He was a specialist certified by both the American Board of Ophthalmology and the American Board of Otolaryngology. He was a Fellow of the American Academy of Ophthalmology and Otolaryngology, and of the American College of Surgeons, and a member of the Pacific Coast Oto-Ophthalmological Society, American Medical Association, Oregon State Medical Society, and Marion-Polk County Medical Society. He also was an active member of the Presbyterian church, the Kiwanis Club, and Chamber of Commerce, and he was a Mason and a Shriner.

On April 26, 1956, Dr. Clement died at the Salem General Hospital, Salem, Oregon, where he had served as a member of the staff. Survivors are his widow, two sons and two daughters.

ROBERT HULL COURTNEY

Dr. Robert Hull Courtney was born March 20, 1893, at Lenoir, North Carolina. He received his early education at Trinity Park Preparatory School, and his premedical education at Trinity College, now Duke University, at Durham, North Carolina. He was graduated from the Medical College of Virginia in 1919. His internship was served at Boston City Hospital. From 1921 to 1926 he was associate professor of physiology and pharmacology at the Medical College of Virginia. In 1924 he became associated with the Department of Ophthalmology. He was appointed professor of ophthalmology in 1938, and clinical professor of ophthalmology in 1952, in which capacity he served until his death.

Dr. Bob, as he was known to his confreres, was a likeable person, a good story teller, a fine teacher, and a real gentleman. His students and residents had great respect for him as a scholar. He, in turn, had great confidence in them.

Dr. Courtney was active in the medical societies to which he belonged. He was president of the Richmond Eye, Ear, Nose and Throat Society in 1936, and of the Virginia Society of Ophthalmology and Otolaryngology in 1951 and 1952. He was a Fellow of the American Academy of Ophthalmology and Otolaryngology, and a member of the American Ophthalmological Society, and of the Association for Research in Ophthalmology. He was on the staff of the Hospital Division of the Medical College of Virginia.

Dr. Courtney died in Richmond, May 23, 1956, at the age of 63. He is survived by his wife, Margaret Ewell Courtney, a son, Robert Hull Courtney, Jr., and a daughter, Mrs. Margaret Gray Kraft.

HERMAN PORTER DAVIDSON

Dr. Herman Porter Davidson was born in Virginia in 1891. Throughout his long professional life he lived in Chicago, and it was there that he died on October 9, 1955.

Having received his degree as Doctor of Medicine from the Johns Hopkins University School of Medicine in 1917, Dr. Davidson served his internship at Johns Hopkins Hospital, and then became house officer at New Haven Hospital, New Haven, Connecticut. He obtained postgraduate training in ophthalmology at the Massachusetts Eye and Ear Infirmary, Boston.

The American Board of Ophthalmology certified Dr. Davidson as a specialist in 1924. He was elected a Fellow of the American Academy of Ophthalmology and Otolaryngology the same year, and attained the status of Life Fellow in 1954. He was also a member of the American Medical Association.

Dr. Davidson served on the staffs of Chicago Memorial Hospital and Presbyterian Hospital, Chicago.

WILLIAM ANTHONY DEFNET

Dr. William A. Defnet was born at Escanaba, Michigan, in 1885. He received his degree in medicine in 1911 from the Detroit College of Medicine, and was then associated with Dr. Burt Shurley in Detroit for many years before going into private practice. He was on the staff of Harper Hospital for over twenty years and served as emeritus professor of his alma mater, which is now the Wayne University College of Medicine.

Dr. Defnet was a Life Fellow of the American Academy of Ophthalmology and Otolaryngology, and a member of the Wayne County Medical Society, the American Medical Association, and the Michigan Medical Association. He also belonged to the Detroit Athletic Club.

Dr. Defnet died February 1, 1956, at Harper Hospital, Detroit. A brother, Dr. Henry J. Defnet of Escanaba, Michigan, survives.

JUSTIN MARTIN DONEGAN

Dr. Justin Martin Donegan died on March 7, 1956, at Presbyterian Hospital, Chicago. At the time of his death, he was chairman of the Department of Ophthalmology of Presbyterian Hospital and associate professor of ophthalmology (Rush) at the University of Illinois College of Medicine. He also served on the staff of St. Joseph's Hospital.

Dr. Donegan was born in Davenport, Iowa, April 11, 1911. His premedical education was obtained at St. Ambrose College. He then attended the State University of Iowa College of Medicine, from which institution he received his medical degree in 1935. He interned at Milwaukee County Hospital in Wauwatosa, Wisconsin, then became a resident in ophthalmology at Albert Merritt Billings Hospital, Chicago, and later at St. Luke's Hospital, Chicago.

Certified as a specialist by the American Board of Ophthalmology in 1941, Dr. Donegan was elected a Fellow of the American Academy of Ophthalmology and Otolaryngology in 1948. He was a member of the American Medical Association, Chicago Medical Society, Chicago Ophthalmological Society, and the Pan American Association of Ophthalmology.

FAYETTE CLAY EWING

Dr. Fayette Clay Ewing was born May 28, 1892, at the Ariel Plantation in La-Fourche Parish, Louisiana. He studied at the University of the South, Sewanee, Tennessee, the University of Mississippi, and Jefferson Medical College of Philadelphia. He served internships at New York City and London hospitals, and practiced ophthalmology and otolaryngology in Washington and St. Louis. In 1947, Jefferson Medical College honored him for having practiced medicine actively for 63 years.

Dr. Ewing was one of the founders, and a Life Fellow, of the American Academy of Ophthalmology and Otolaryngology. He was a member of Great Britain's Royal Society of Medicine and a fellow of the American College of Surgeons and of the American Medical Association. He served in World War I as major in the Medical Reserve Corps at Camp Beauregard, and was chief of the Department of Eye, Ear, Nose and Throat at the Veterans' Rehabilitation Hospital, Alexandria, Louisiana, from 1919 to 1926.

Dr. Ewing has been known throughout the world as "the man who made the little shaggy Scotch Terrier popular." He wrote "The Book of the Scottish Terrier," a standard work on the dog in the United States and Great Britain. Most dog experts consider him the first Scotch Terrier breeder and promoter in Ameri-

ca. In 1900 he was a joint founder of the Scottish Terrier Club of America. His plantation's Nosegay Kennels were famous throughout the world. The Scottish Terrier Club of Scotland made him honorary president, the only American who has received this honor.

Dr. Ewing was a noted Shakespearean writer and lecturer. He published "Hamlet, an Analytical and Psychological Study" in 1934.

On April 15, 1956, Dr. Ewing died at his Louisiana plantation, at the age of 93. Survivors are his wife, Rowena A. Clarke Ewing; a son, Donald M. Ewing, who is associate editor of the Shreveport Times; four grandchildren, and several great grandchildren.

HENRY JOSEPH FEASTER

Dr. Henry Joseph Feaster was born in New York City, May 30, 1893. He died in Brooklyn, March 8, 1956.

Dr. Feaster attended the College of the City of New York for two years and then entered the Long Island College Hospital of Medicine, which granted his degree of Doctor of Medicine in 1916. His internship was served at Long Island College Hospital.

For postgraduate work, Dr. Feaster went to Harvard Medical School in 1918. He also studied bronchoesophagology at the Pennsylvania University School of Medicine, and otorhinolaryngology at the University of Vienna. He saw service in World War I.

Returning to Brooklyn to establish his home and medical practice, Dr. Feaster became associated with his alma mater, the name of which was changed first to Long Island College of Medicine and then in 1950 to State University of New York College of Medicine. At his death, he was assistant professor of clinical otolaryngology in that school. He was also attending otolaryngologist at Long Island College, Victor Memorial, and Bay Ridge hospitals, Norwegian Lutheran Deaconess Home and Hospital, and Angel Guardian Home.

Dr. Feaster was certified as a specialist by the American Board of Otolaryngology in 1930. He was a Fellow of the American Academy of Ophthalmology and Otolaryngology, of the American College of Surgeons, and of the American Academy of Compensation Medicine. A past president of the Bay Ridge Medical Society, he was also a member of the Medical Society of the State of New York, the Medical Society of Bay Ridge, and the American Medical Society of Vienna.

NATHANIEL ARTHUR FISCHER

Dr. Nathaniel A. Fischer was born April 8, 1886, at Bradford, Pennsylvania. He attended Central High School of Pittsburgh, and then entered the University of Pittsburgh School of Medicine, where he received his degree as Doctor of Medicine. He was a specialist certified by the American Board of Otolaryngology, and began practicing in Pittsburgh in 1910. He was clinical professor emeritus of otology at his alma mater, senior staff consultant at the Eye and Ear Hospital and at St. Francis Hospital of Pittsburgh, and consultant in ear, nose and throat diseases for the U. S. Marine Hospital, Pittsburgh.

Dr. Fischer was a Fellow of the American Academy of Ophthalmology and Otolaryngology and of the American College of Surgeons. He also held membership in the American Medical Association, the Allegheny County Medical Society, the Pennsylvania Medical Society, and the Pittsburgh Otological Society. He was past president of the Pennsylvania Academy of Ophthalmology and Otolaryngology.

Dr. Fischer died July 28, 1955, at the age of 69, at the W. A. Foote Memorial Hospital, Jackson, Michigan. Survivors are his wife, Lorraine S. Fischer; a son, Dr. Arthur J. Fischer, who is also a Fellow of the Academy; three daughters, and nine grandchildren.

JAMES LAWRENCE FLYNN

Dr. James L. Flynn was born May 8, 1910, at Boston, Massachusetts. He graduated from Boston Latin School and received his Bachelor of Arts degree from Boston College, cum laude, in 1934. In 1939 he received his degree as Doctor of Medicine from Georgetown University School of Medicine. From 1939 to 1940 he interned at St. Vincent's Hospital, Bridgeport, Connecticut. He was house officer on the pediatric service at Boston City Hospital in 1940 and 1941 and a resident in ophthalmology and otolaryngology at Boston City Hospital in 1943 and 1944. On July 1, 1944 Dr. Flynn began private practice in Boston.

He became assistant visiting ophthalmic surgeon at Boston City Hospital, and at Carney Hospital; assistant visiting ear, nose and throat surgeon at St. Elizabeth's Hospital; ophthalmology consultant at Leonard Morse Hospital, and ophthalmic visiting surgeon at Newton-Wellesley Hospital. In 1951 and 1952 he completed two parts of the Lancaster Ophthalmic Graduate Study Courses.

Dr. Flynn was a Fellow of the American Academy of Ophthalmology and Otolaryngology, a member of the American Medical Association, the New England Ophthalmological Society, the Massachusetts Medical Society, and a diplomate of the American Board of Ophthalmology. He was a member of St. Patrick's Church, Natick, Massachusetts.

Dr. Flynn was extremely interested in the Boston College Alumni, St. Luke's Guild, the Camillus Club, and the Holy Name Society.

The third week in September 1955, Dr. Flynn contracted bulbar poliomyelitis and died within a week from this disease. He will be remembered by his associates and many friends as an exceeding kind and gentle person. Survivors are his wife, Alice G. (Allen) Flynn, a son, James L., Jr., and a daughter, Alice P. Flynn.

JONAS S. FRIEDENWALD
1897-1955

(See Eulogy in the TRANSACTIONS, November-December 1955, p. 798.)

ARTHUR VARNEY GARLOCK

Dr. Arthur V. Garlock was 71 years of age on September 22, 1955. For thirty-nine years he practiced medicine as an ophthalmologist and otolaryngologist, in the same offices, in Bemidji, Minnesota. He had just purchased the Bemidji Gas Company Building, where new, ground-floor offices were being prepared for him; but these quarters will not be occupied by Dr. Garlock, for on June 29, 1956, he died of a heart attack, after once more climbing that long flight of stairs to his old office.

A long career of public service and participation in civic organizations, besides his medical practice and his love of music, art, and farming, made his life a full and happy one.

In 1921 and 1922 Dr. Garlock was mayor of Bemidji, and later he served for five years as president of the Bemidji park board. He was a member of the board of trustees of the First Methodist church, past master of the Bemidji Masonic lodge, past commander of the Knights Templar, past exalted ruler of the Elks lodge, past patron of the Order of Eastern Star, and past noble grand of the Independent Order of Odd Fellows.

Dr. Garlock received his degree in medicine at Northwestern University Medical School, Chicago, in 1910. He was married that same year. He then spent four years in general practice and one year in teaching before beginning his practice in Bemidji. He was a member of the American Academy of Ophthalmology and Otolaryngology, of the Upper Mississippi branch of the American Medical Association, and of the Minnesota State Medical Association.

Dr. Garlock was well known as a fine violinist and shared his talent by participating in many public functions. He had been painting landscapes and botanical subjects for many years. Another profound interest was in agriculture. He had founded one of the most outstanding herds of Guernsey cattle, and his farm is truly a "showplace" in the state of Minnesota. Survivors, in addition to his wife, Mabel Garlock, are a son, Robert, a daughter, Mrs. Edward Haskell, a brother, Dr. D. H. Garlock of Los Angeles, and several other brothers and sisters.

JOHN RANDOLPH GILLUM

Dr. John Randolph Gillum was born in Rockville, Indiana, in 1878. His early education was obtained at Virginia Military Institute, at Wabash College, and at the University of Indiana. Medical training was secured at Jefferson Medical College of Philadelphia, where he received the degree of Doctor of Medicine in 1904. He served an eight-months' internship at Easton Hospital, Easton, Pennsylvania, and then went to Philadelphia General Hospital as an intern for one year. He studied in Vienna, Berlin, and elsewhere abroad.

In 1906 Dr. Gillum established his home and medical practice in Terre Haute, Indiana, and became a member of the staff of Union Hospital.

Dr. Gillum was certified as a specialist by the American Board of Otolaryngology. In 1920 he was elected a Fellow of the American Academy of Ophthalmology and Otolaryngology and was elevated to Life Fellowship in 1950, after thirty years of continuous membership. In October, 1954, Dr. Gillum was presented the Fifty-Year Pin of the Indiana State Medical Association. He was one of the founders of the Associated Physicians and Surgeons Clinic, a member of the American Medical Association, past president of the Indiana Academy of Ophthalmology and Otolaryngology, and a member of Indiana State Medical Association, Terre Haute Academy of Medicine, Vigo County Medical Society, and the Aesculapian Society of Wabash Valley.

Dr. Gillum was a director of the Indiana State Bank, a member of Terre Haute Chapter No. 11, Royal Arch Masons, life member of Zorah Shrine Temple, and a member of the First Congregational Church, Terre Haute. He was very active in local music organizations.

Dr. Gillum died March 30, 1956, at Terre Haute, at the age of 78. His wife's death occurred one month later.

JOHN ELWIN GLEASON

Dr. John Elwin Gleason was born October 16, 1878, at Oxford, New York. He was educated at Yale University, and later attended the University of Michigan Medical School, Ann Arbor, where he received his degree as Doctor of Medicine in 1903.

Dr. Gleason was on the staffs of Grace Hospital and Wayne University Medical School, both in Detroit, Michigan. He was a fellow of the American College of Surgeons, a specialist certified by the American Board of Otolaryngology, and a member of the American Academy of Ophthalmology and Otolaryngology. He also was a member of the Wayne County Medical Society, Michigan State Medical Society, Detroit Ophthalmological Society, and Detroit Ophthalmological Club.

Dr. Gleason was active in golf and was a member of the Detroit Golf Club.

Surviving Dr. Gleason are a daughter, Mrs. Gerald Richardson, Fort Lauderdale, Florida, and two grandchildren.

EDWARD CRESCENTUS GOLDCAMP

Dr. Edward Crescentus Goldcamp was born on June 27, 1890, at Ironton, Ohio, where he lived until his graduation from Ironton High School. In 1914 he graduated from Jefferson Medical College, Philadelphia. By 1919 Dr. Goldcamp decided to restrict his practice to diseases of the ear, nose and throat, and began to practice at Warren, Ohio.

Dr. Goldcamp later established his home and practice at Youngstown, Ohio. He served on the teaching staff of the nurses' training school. He was in charge of the bronchoscopic service, and was visiting physician in the ear, nose and throat service, soon becoming senior staff member, at Youngstown City Hospital. He also was a member of the staff of Warren City Hospital. He is known as a pioneer in bronchoscopy in northeastern Ohio and achieved outstanding success in this field.

Dr. Goldcamp served his country in World War I, was an active member in St. Edwards Catholic Church, and participated in various musical organizations. He was interested in sports all his life.

Certified as a specialist by the American Board of Otolaryngology in 1932, Dr. Goldcamp was elected a Fellow of American Academy of Ophthalmology and Otolaryngology that same year. He was a member of Mahoning County Medical Society, Ohio State Medical Society, American Medical Association, Pittsburgh Otological Society, Youngstown Medical Arts Club, and the Cleveland Academy of Medicine.

On June 2, 1956, Dr. Goldcamp died at Youngstown, Ohio, at the age of 65. He is survived by three brothers: Dr. John S. Goldcamp, who is a Fellow of the American Academy of Ophthalmology and Otolaryngology, and Dr. S. W. Goldcamp, both of Youngstown, Ohio, and Cyril F. Goldcamp of Pittsburgh, Pennsylvania; and one sister, Hilda M. Weinfurtner of Pompano Beach, Florida.

A. MILTON GOLDMAN

Dr. A. Milton Goldman was born November 1, 1891, in Kansas City, Missouri. Here he attended public schools and later studied at the University of Kansas, where he received his Bachelor of Science degree. He graduated from Har-

vard Medical School in 1917, then spent his internship at the Boston City Hospital and at Childrens' Hospital in Boston. After two years of general practice in Kansas City, Missouri, he decided to limit his practice to ophthalmology. He moved back to the east, making Rockville Centre, New York, his home. Six months was spent as resident at the Massachusetts Eye and Ear Infirmary. Dr. Goldman became the protege and associate of Dr. Hornbuglar, a leading ophthalmologist of Brooklyn. Later Dr. Goldman opened offices in New York City and in Rockville Centre, but when the local practice became too large, he closed his New York City office and practiced only in Rockville Centre, continuing until the middle of 1954.

For over nine years Dr. Goldman suffered from cancer, but he continued his practice and served as a leading Long Island ophthalmologist until a year before his death, August 26, 1955. He was a man of great courage. A great deal of time was spent with his family, and often they and friends would gather around the piano to listen to the original renditions of popular music that Dr. Goldman loved to play. When on vacation, he became an avid fisherman and camping companion of his brother George.

Dr. Goldman spent much time serving his profession and various organizations. He was consulting ophthalmologist at Nassau Hospital, Mineola; Mercy Hospital, Rockville Centre; South Nassau Hospital, Oceanside; Mary Immaculate Hospital, Jamaica; and director of ophthalmology at Meadowbrook Hospital, East Meadow. He was a Senior Fellow of the American Academy of Ophthalmology and Otolaryngology and a member of the New York Academy of Medicine. He was president of the board at Mary Immaculate Hospital, and one of the founders and past presidents of the Nassau County Society of Ophthalmology, and he served in many civic organizations.

In 1920 Dr. Goldman married Caroline Elias. She survives with two children and five grandchildren.

OLIN MANSELL GOODWIN

June 21, 1915, was the birth date of Olin Mansell Goodwin. His early education was obtained in his native city, Mannington, West Virginia. He then attended West Virginia Wesleyan College. In 1941 he received his degree in medicine from the Medical College of Virginia, Richmond, Virginia. He interned at the University of Virginia Hospital in Charlottesville, and was a resident in otolaryngology for three years at the University of Iowa, Iowa City, Iowa. In 1950 he began private practice in Gallaway, West Virginia.

A specialist certified by the American Board of Otolaryngology, Dr. Goodwin was a Fellow of the American Academy of Ophthalmology and Otolaryngology; a fellow of the American College of Surgeons; and a member of the West Virginia State Medical Society and of the Marion County Medical Society.

During World War II, Dr. Goodwin served in the United States Air Corp as a flight surgeon. He was a member of the official board of the First Methodist Church. Refinishing the antique furniture which he collected, family projects, and the sport of fishing were other activities in which Dr. Goodwin spent much time.

On February 28, 1956, Dr. Goodwin died of myocardial infarction, at the age of 40 years. Surviving him are his wife, Helen, daughters, Nancy and Sarabeth, and a son, Bill.

HENRY COOPER HADEN

Dr. Henry Cooper Haden was born on December 7, 1873, at Galveston, Texas. He attended the University of Pennsylvania Department of Medicine, Philadelphia, receiving the degree of Doctor of Medicine in 1895.

A specialist certified by the American Board of Otolaryngology, Dr. Haden was elected to fellowship in the American Academy of Ophthalmology and Otolaryngology in 1913. He was elevated to the status of Life Fellow in 1943, having been a member of the Academy continuously for thirty years.

Dr. Haden maintained his home and office in Houston, Texas. He was a member and past president of the Harris County Medical Society; a member and past president of the American Ophthalmological Society; a member of the American Medical Association; and a fellow of the American College of Surgeons.

At one time Dr. Haden was head of the department of ophthalmology and

otolaryngology at the University of Texas School of Medicine in Galveston. For many years, he was affiliated with the Hermann Hospital, Houston, and he was subsequently named honorary ophthalmologist of that institution. He served in the Armed Forces during World War I.

Dr. Haden died in Houston on April 24, 1956, at the age of 82 years.

EDWIN PERRY HALL

Dr. Edwin Perry Hall was born in 1880, and died in Oneonta, New York, where he had made his home for many years, on April 9, 1956.

A graduate of Hahnemann Medical College and Hospital of Philadelphia, he received his medical degree in 1903. In 1919 he decided to limit his practice to diseases of the ear, nose and throat, and he was certified in that specialty by the American Board of Otolaryngology in 1925.

The American Academy of Ophthalmology and Otolaryngology elected Dr. Hall a Fellow in 1928. He was a Senior Fellow of the Academy at the time of his death. He had been a member of the American Medical Association, New York State Medical Society, New York Homeopathic Society, and the American Institute of Homeopathy; and he was a member and past president of the Otsego County Medical Society.

The hospital and teaching appointments which Dr. Hall held during his professional career included attending otolaryngologist at the General Hospital, St. Joseph's Hospital, and City Hospital for Contagious Diseases; and clinical instructor in otolaryngology at the Syracuse Free Dispensary of Syracuse University. He was a member of the staff of the Amelia Fox Memorial Hospital, Oneonta.

LAMAR BLEWETT HARPER

Dr. LaMar Blewett Harper entered the United States Navy in 1938 and retired January 1, 1946, with the rank of Lieutenant Commander.

A graduate of the George Washington University School of Medicine, Washington, D. C., in 1936, Dr. Harper found his major interest in diseases of the eye. Following his retirement from the Navy, he was certified as a specialist by the American Board of Ophthalmology in 1952.

Dr. Harper was elected a Fellow of the American Academy of Ophthalmology and Otolaryngology in 1954. He was also a member of the American Medical Association, San Luis Obispo County Medical Society, and the California Medical Association.

Dr. Harper practiced his profession, as a civilian, in San Luis Obispo, California, and was on the staff of French Hospital, San Luis Obispo, and War Memorial Hospital, Paso Robles. He later moved to Yuma, Arizona, and it was there that he died on May 2, 1956, at the age of 43 years.

Three children survive Dr. Harper.

CHARLES MORGAN HARRIS, JR.

Dr. Charles Morgan Harris was born in Montgomery, Alabama, in 1897 and died in Jersey City, New Jersey, on March 9, 1956.

After receiving his preliminary education at Talledega College in Alabama and at Boston University, Dr. Harris attended Howard University College of Medicine, Washington, D. C. He received his degree of Doctor of Medicine from the latter institution in 1924.

Dr. Harris was certified as a specialist by the American Board of Otolaryngology in 1936 and elected a Fellow of the American Academy of Ophthalmology and Otolaryngology in 1949. He was a member of the American Medical Association, Hudson County Medical Society, Philadelphia County Medical Society and the Medical Society of the State of Pennsylvania, and a fellow of the Academy of Medicine of Northern New Jersey.

During his professional life, Dr. Harris served on the staffs of the Jersey City Medical Center, Community Hospital, Newark; and as chief of otolaryngology and endoscopy at Mercy Douglas Hospital, Philadelphia. He was a member of the courtesy staff of Sydenham Hospital, New York City. He maintained offices in both Jersey City and Philadelphia.

Dr. Harris is survived by his wife, who is also a physician.

MARTIN LUTHER HARSHMAN

Dr. Martin Luther Harshman was born on August 19, 1913, at Frankfort, Indiana. He attended elementary school and high school in Mulberry, Indiana. For three years he attended Purdue University at Lafayette. He received his Bachelor of Science degree in 1937 and his degree as Doctor of Medicine in 1939 at the University of Indiana. He interned at St. Elizabeth Hospital in Lafayette, and at St. Vincent's Hospital in Indianapolis. He then began private practice at Colfax, Indiana. Postgraduate work in otolaryngology was done at Washington University, St. Louis, Missouri.

Dr. Harshman was on the staffs of St. Elizabeth Hospital, and the Lafayette Home Hospital, both of Lafayette. He was a member of the Tippecanoe County Medical Society, Indiana State Medical Society, American Medical Association, Indiana Academy of Ophthalmology and Otolaryngology, American College of Allergists, and the American Society of Ophthalmologic and Otolaryngologic Allergy, as well as being a Fellow of the American Academy of Ophthalmology and Otolaryngology. He was a specialist certified by the American Board of Otolaryngology.

In 1942 Dr. Harshman entered military service as a Captain in the United States Army Medical Corps. He served at Camp Grant and was stationed at the Presidio in California, and in the Pacific Theatre of Operations. He was separated from the service as a Major in the Medical Corps. In 1947 he became otolaryngologist at Arnett Clinic, Lafayette.

Other interests, besides his specialty, were in photography, fishing, and camping.

On October 19, 1955, Dr. Harshman died, at the age of 42. He is survived by his wife, Lucille; his mother, Mrs. Laura Harshman; one brother and one sister.

BENJAMIN FRANKLIN HODSDON

Dr. Benjamin Franklin Hodsdon of Jacksonville, Florida, died May 29, 1956, following a heart attack. He was 90 years of age.

A native of Maine, Dr. Hodsdon was born at Berwick in 1865 and took great pride in his New England heritage. He worked his way through college and was graduated in 1897 from Bowdoin Medical School, then in turn from the New York Polyclinic, the Chicago Eye, Ear, Nose and Throat College, and in 1908, from the University of Illinois College of Medicine. He also studied in France, and frequently during the summer months engaged in postgraduate study.

In 1910 Dr. Hodsdon was licensed to practice in Florida. In 1911 he came to Miami from Manchester, Maine, seeking relief from asthma. He remained in Miami for thirty years and became the city's first eye, ear, nose and throat specialist. For twenty-five years he was a member and twice president of the Dade County Board of Health, and for a number of years was the local eye, ear, nose and throat surgeon for the Florida East Coast Railroad. He and the late Dr. James M. Jackson selected the site of the present Jackson Memorial Hospital where he served on the staff. Locally he was a Mason, Shriner, Rotarian, Elk, Odd Fellow, and a member of the Central Baptist Church. In 1940 he moved to Tallahassee, where he resided for five years before coming to Jacksonville to make his home. In Jacksonville he held membership in the First Baptist Church.

Dr. Hodsdon was a Life Member of the Florida Medical Association, holding honorary status since his retirement; he had been a member for forty-four years. In 1950 he received a gold medal from the New Hampshire Medical Society for fifty years' membership. He also was a member of the Vermont State Medical Society, and the American Medical Association, and he was a Life Fellow of the American Academy of Ophthalmology and Otolaryngology.

Survivors include a daughter, Mrs. Bryan Page, of Miami, and two grandchildren.

JOSEPH STANTON HORY

Dr. Joseph Stanton Hory was born on May 10, 1895, in Girardsville, Pennsylvania. He died on January 1, 1956, in New York City, where he had been engaged in the practice of medicine for all of his professional life.

A graduate, in 1919, of Fordham University School of Medicine, New York City, Dr. Hory had restricted his practice to diseases of the eye since 1923. He was certified by the American Board of Ophthalmology in 1934.

For many years Dr. Hory held staff appointments at Morrisonia, Seton and Union hospitals. He also served on the staff of Foundling Hospital, Bronx Eye and Ear Infirmary, and Manhattan Eye, Ear, and Throat Hospital. He was assistant clinical professor of ophthalmology at New York Medical College, Flower and Fifth Avenue hospitals.

Dr. Hory was elected a Fellow of the American Academy of Ophthalmology and Otolaryngology in 1939. He was a fellow of the American College of Surgeons and of the New York Academy of Medicine, and a member of the American Medical Association, American Medical Association of Vienna, Bronx Medical Association, and Bronx County Medical Society.

WILLIAM FRANKLIN HUGHES

Dr. William Franklin Hughes, a Life Fellow of the American Academy of Ophthalmology and Otolaryngology, died in Indianapolis, Indiana, on April 19, 1956.

Dr. Hughes, who was born in Owen County, Indiana, on October 11, 1871, received his undergraduate education at Indiana State Teachers College. He attended the Medical College of Indiana at Indianapolis, where he received his degree as Doctor of Medicine in 1902. Postgraduate work was done at Moorfields Hospital in London, and under the elder Fuchs in Vienna.

In 1903, Dr. Hughes began the practice of medicine in Indianapolis, where he was on the staff of Indiana University Hospitals and Indianapolis General, Methodist, St. Vincent's, and St. Francis' hospitals. Early in the century, he became affiliated with the Indiana University School of Medicine, where he was an outstanding teacher in ophthalmology. In 1933, he became chairman of the department of ophthalmology, and became professor emeritus upon his retirement in 1943.

In addition to his Academy membership, Dr. Hughes was a member of the Indiana Academy of Ophthalmology and Otolaryngology and of the Indianapolis Ophthalmological and Otolaryngological Society. He was an associate member of the American Medical Association and was affiliated with state and local medical societies.

For many years Dr. Hughes maintained a large apple orchard at his childhood home, and he was considered a skillful horticulturist. In recent years, he spent several months each winter in Florida and became an ardent fisherman.

Dr. Hughes is survived by his wife, Alta Rentschler Hughes; a daughter, Mrs. John R. Swan; and two sons, Dr. Richard R. Hughes, and Dr. William F. Hughes, Jr., who is also a Fellow of the Academy.

DAVID NATHANIEL HUSIK

David N. Husik was born on August 15, 1883. He attended the lower Philadelphia Public Grade schools and was graduated from Central High School in 1903, and from the University of Pennsylvania School of Medicine in 1907. He interned at the Philadelphia General Hospital from 1907 until 1908, and then began private practice.

Dr. Husik was chief of the ear, nose and throat department and assistant professor of laryngology at the University of Pennsylvania Hospital until 1928. He was chief of the ear, nose and throat department of the Philadelphia General Hospital for thirty-five years, and became an active consultant there in 1948. He was consultant to the Police and Fire Departments for many years. He served as associate professor of otolaryngology at the graduate school of the University of Pennsylvania. He was also instructor in otology and laryngology at the University of Pennsylvania, the Philadelphia Polyclinic, and the Postgraduate School of the University of Pennsylvania; in addition, he was surgeon in charge of the nose and throat dispensary of the University Hospital for many years.

Dr. Husik was a Life Fellow of the American Academy of Ophthalmology and Otolaryngology, of the College of Physicians, and of the Philadelphia County Medical Society; a senior fellow of the American Laryngological, Rhinological and Otological Society; a life member of the Medical Club; a fellow of the Philadelphia Laryngological Society, and a member of the American Medical Association.

Dr. Husik was a Life Fellow of the papers, the last of which was entitled "Sclerotic Mastoiditis and Intracranial Complications." He was highly esteemed and his work was a valuable contribution to his specialty.

Dr. Husik died at the University Hospital on November 20, 1955. He is survived by his wife, Marguerite, and a daughter, Antoinette.

FRANK JOSEPH HYNES

Dr. Frank Joseph Hynes was born on October 16, 1908, in New York City, and died in that city on June 7, 1956.

Having obtained the degree of Bachelor of Science in 1929 at the College of the City of New York, Dr. Hynes entered New York University. He received his medical degree in 1933 from University and Bellevue Hospital Medical College.

In 1938 Dr. Hynes decided to restrict his practice to diseases of the ear, nose and throat, and he was certified as a specialist by the American Board of Otolaryngology in 1940.

The American Academy of Ophthalmology and Otolaryngology elected Dr. Hynes a Fellow in 1941. Other medical organizations of which he was a member are the American Laryngological, Rhinological and Otological Society, Medical Society of the County of New York, Medical Society of the State of New York, New York Celtic Medical Society, American Trudeau Society, and American College of Allergists. He was a fellow of the International College of Surgeons and the American College of Surgeons.

Dr. Hynes served on the staffs of Manhattan Eye, Ear and Throat Hospital and New York Hospital.

MARTIN JOHN JOYNT

Dr. Martin John Joynt died at the age of 77 at the Sacred Heart Hospital in LeMars, Iowa, on December 7, 1955.

In 1905 Dr. Joynt graduated from the State University of Iowa College of Medicine, Iowa City, Iowa, where he received the degrees of bachelor of science and Doctor of Medicine. He joined the staff of the Sacred Heart Hospital, where he also was instructor of eye, ear, nose and throat diseases at the Sacred Heart School of Nursing.

Dr. Joynt was a member of the Plymouth County Medical Society, Iowa State Medical Society, and Sioux Valley Eye and Ear Academy, and was a Senior Fellow of the American Academy of Ophthalmology and Otolaryngology.

JOHN HENRY KINCAID

Dr. John Henry Kincaid was born in 1871 at Fincastle, Campbell County, Tennessee. He attended the University of Tennessee and received his degree as Doctor of Medicine from the University of Michigan Medical School. At Knoxville, Tennessee, about 1895, Dr. Kincaid began his private practice in ophthalmology and otolaryngology.

Dr. Kincaid was a Life Fellow of the American Academy of Ophthalmology and Otolaryngology, and a member of the American Medical Association, American College of Surgeons, and of the International College of Surgeons. He also had a great love for grand opera and all other types of music, as well as art.

On January 20, 1955, at the age of 84, Dr. Kincaid died in Brownsville, Tennessee. Survivors are two nieces and one nephew.

EDWARD DANIEL KING

Dr. Edward D. King was born September 20, 1888, at Covington, Kentucky. He attended Xavier University, receiving his Bachelor of Arts degree in 1908. In 1912 he received his degree as Doctor of Medicine at the University of Cincinnati College of Medicine.

From 1912 to 1945 Dr. King was in private practice in Cincinnati, Ohio. He then moved to Hollywood, California, where he practiced his specialty as an otolaryngologist. Postgraduate work was done at Good Samaritan Hospital, and General Hospital, both in Cincinnati.

Dr. King was especially interested in allergy and plastic surgery of the nose and face. He published many articles on this subject. He was clinical professor of otolaryngology at College of Medical Evangelists at Loma Linda and Los Angeles; a former member of the faculty of his alma mater; a member of the American Society of Plastic and Reconstructive Surgery, American Academy of Ophthalmology and Otolaryngology, American Laryngological Association, and the American Laryngological, Rhinological and Otological Society; a fellow of the American College of Surgeons; past-president of the Cincinnati Academy of Medicine; past-president of the Cincinnati League for the Hard of Hearing; and a member of many civic organizations. Dr. King also served on the staffs

of the Good Samaritan, Cincinnati General, and Children's hospitals in Cincinnati; St. Joseph's Hospital in Burbank, and the Presbyterian, Olmsted Memorial, and Los Angeles County hospitals in Los Angeles.

On January 15, 1956, Dr. King died at the age of 67. He will long be remembered as a man of dignity with an agreeable personality, and as a physician and friend who was modest and kind. He will be thought of as a man who loved music, and, although he played no instrument himself, as one who contributed greatly to the Philharmonic and Hollywood Bowl concerts. Dr. King is survived by his wife, Genevieve, and three sons and six grandchildren.

ARNOLD HERMAN KNAPP

Born in New York on August 20, 1869, the son of Dr. Herman Jakob Knapp, Arnold Herman Knapp was reared in a scientific atmosphere. His father had resigned the professorship of ophthalmology at the University of Heidelberg in 1868 to come to New York, where he established the New York Ophthalmic and Aural Institute.

After obtaining his academic degree at Harvard University in 1889, Dr. Arnold Knapp returned to New York for his medical training at the College of Physicians and Surgeons of Columbia University. A surgical internship at Roosevelt Hospital was followed by ophthalmic training in European clinics. At the New York Ophthalmic and Aural Institute, Dr. Herman Knapp and his son, Dr. Arnold Knapp, worked together assiduously—often with divergent views but always with a common desire for scientific truth and intellectual honesty. When the father retired in 1909, new quarters were established and a new name, The Herman Knapp Memorial Eye Hospital, was given the institute. Here Dr. Arnold Knapp continued his clinical activities and postgraduate instruction until 1940, when this institution was merged with the Institute of Ophthalmology of Presbyterian Hospital. With the assets of the Herman Knapp Memorial Eye Hospital, a Knapp Memorial Foundation was established under the auspices of Columbia University for the advancement of postgraduate study, teaching, and research in ophthalmology. The continuation of this Foundation will serve as a living monument to Dr. Arnold Knapp, whose life was dedicated to the advancement of ophthalmology.

In 1911 Dr. Knapp assumed the editorship of the Archives of Ophthalmology, for which he was eminently fitted. For thirty-eight years he guided the destinies of this magazine. In 1917 he published a volume on "Medical Ophthalmology."

Dr. Arnold Knapp was professor of ophthalmology at Columbia University for twenty-five years, and became professor emeritus—a position he held until his death. In recognition of his outstanding achievements, Columbia University awarded him an honorary degree of Doctor of Science in 1931, and in 1953, on the occasion of the twenty-fifth anniversary of the founding of the Columbia Presbyterian Medical Center, he received a Distinguished Service Award. The American Ophthalmological Society bestowed upon him its Lucien Howe Medal for Meritorious Services, and the National Society for the Prevention of Blindness, its Leslie Dana Gold Medal. In 1946 he became the second American to deliver the Bowman Lecture before the Ophthalmological Society of the United Kingdom, and he was elected to honorary membership in that distinguished society. He also was an honor member of the Societe Francaise d' ophtalmologie and of the Deutsche Ophthalmologische Gesellschaft. These are but a few of the honors he attained.

Dr. Knapp and the late Dr. Derrick Vail, Sr., won great acclaim as surgeons in performing the intracapsular method of cataract extraction. Although his life was dedicated to ophthalmology, Dr. Knapp maintained a great interest in art and was an avid reader of the classics.

On February 29, at the age of 86, Dr. Arnold Knapp died of arteriosclerotic heart disease. Ophthalmology has lost one of its most illustrious members. We shall remember him as a clear-thinking, forthright gentleman, who by his long and fruitful labors so greatly enriched the specialty of ophthalmology. He is survived by four children: Miss Elizabeth Knapp, Mrs. Ellen Morandiere, John A. Knapp, and Dr. Philip Knapp, who is a member of the staff of the Institute of Ophthalmology of the Presbyterian Hospital in New York City and is also a Fellow of the Academy.

ADOLPH KREBS

In 1913 Dr. Adolph Krebs became a Junior Member of the American Academy of Ophthalmology and Otolaryngology, in 1938 a Senior Member, and in 1943 a Life Member.

Dr. Krebs had practiced medicine in Pittsburgh, Pennsylvania, for several years before moving to San Diego, California. He opened offices at LaMesa, California, where he practiced ophthalmology for many years.

Dr. Krebs died August 23, 1955, of coronary thrombosis, at the age of 83.

ROBERT SCOTT LAMB

Dr. Robert Scott Lamb was born on October 15, 1876, in Washington, D. C., the son of Dr. Daniel S. Lamb, a famous physician and teacher at Howard University Medical School for many years. The elder Dr. Lamb was a most diligent medical historian of the medical society of the District of Columbia, covering the years 1817 to 1909.

Dr. Robert Lamb received a degree in engineering from Cornell University in 1894, and his degree as Doctor of Medicine from Howard University in 1898. In the same year he began the practice of medicine in Washington, where he was on the staff of the Episcopal Eye, Ear and Throat Hospital.

Dr. Lamb had been a member of the American Academy of Ophthalmology and Otolaryngology for 50 years and was a Life Fellow. He was also a fellow of the American College of Surgeons, and a member of the American Medical Association, the Southern Medical Association, and the Medical Society of the District of Columbia. Dr. Lamb was president emeritus of the Social Hygiene Society of the District of Columbia, and held offices in a number of civic organizations.

Dr. Lamb died on March 4, 1956, in his native city.

JAMES WILLIAM LEHAN

Dr. James William Lehan, who was born in Dunlap, Iowa, on December 9, 1874, died in Greeley, Colorado, on October 30, 1955.

The medical education of Dr. Lehan was obtained at Rush Medical College, Chicago, where he received his degree of Doctor of Medicine in 1897. He also held a degree in pharmacy from the University of Iowa. Dr. Lehan did postgraduate work at the Mayo Clinic, and in Vienna and London.

Dr. Lehan began the practice of medicine in Alexander, Iowa, immediately upon receipt of his degree in 1897, but after five years he returned to his native city, where he remained until 1910. At that time he moved to Greeley, Colorado, where he practiced until his retirement in 1950.

Dr. Lehan was a Life Fellow of the American Academy of Ophthalmology and Otolaryngology, and a member of the American Medical Association, the Colorado Ophthalmological Society, the Colorado Otolaryngological Society, and local medical societies.

Dr. Lehan is survived by his wife, three sons, and a daughter.

HARRY SUMNER LESTER

Dr. Harry Sumner Lester was born in Ciola, Iowa, on March 23, 1871.

Dr. Lester received his undergraduate education at Western Normal College, Shenandoah, Iowa. He received his degree as Doctor of Medicine from the State University of Iowa College of Medicine in 1898, and did postgraduate work at the Chicago Polyclinic Hospital. He began the practice of medicine at Woodford, Wisconsin, and later he moved to Streator, Illinois, where he was on the staff of St. Mary's Hospital.

Dr. Lester was a Life Fellow of the American Academy of Ophthalmology and Otolaryngology, and a member of state and local medical societies. He served as mayor of Streator in 1920 and 1921, and was coroner of La Salle County from 1932 to 1940. He helped to organize the YMCA in Streator, and he was active in many civic and social organizations.

Dr. Lester died on June 24, 1955, in the Veterans Administration Hospital at Dwight, Illinois. He is survived by a son, Dr. Paul J. Lester, and a stepdaughter, Mrs. Leila H. Simons.

BERT G. LEVIN

Dr. Bert G. Levin, a Junior Fellow of the American Academy of Ophthalmology and Otolaryngology, died in St. Paul, Minnesota, on February 8, 1956.

Dr. Levin was born in St. James, Minnesota, on October 31, 1899, and received his early education in that city. His degree as Doctor of Medicine was obtained from the University of Minnesota Medical School in 1924, and he did postgraduate work at the same institution preparing him in the specialties of ophthalmology and otolaryngology. He has been associated with the Earl Clinic in St. Paul for many years and was on the staffs of Midway, Mounds Park, and Children's hospitals at the time of his death.

Dr. Levin was a member of the American Medical Association and the Ramsey County Medical Society, and a Fellow of the American College of Surgeons. He was a board member of Mount Zion Temple and a past president of B'nai B'rith.

Dr. Levin is survived by his wife, Pearl, and two daughters, Susan and Jane.

FRANK E. MALLON

Dr. Frank E. Mallon was born June 18, 1895, in Brooklyn, New York, and was a native of that city all of his life. He attended high school there, and after graduation became an expert stenographer. He graduated from Long Island College of Medicine in 1919, at which time his attention and interest tended toward ophthalmology. By 1932 he was devoting all his professional time to this specialty.

Dr. Mallon served his country in World War I and was in the Medical Reserves during World War II. He was chief of the eye department at Long Island College Hospital, consulting surgeon at St. Mary's Hospital, and he was on the staff of the Brooklyn Eye and Ear Hospital, where he was attending surgeon, the rank he held until his death. He also was instructor of ophthalmology at Long Island College of Medicine.

Dr. Mallon was a Fellow of the American Academy of Ophthalmology and Otolaryngology, and a member of the Medical Society of the County of Kings and of the American Medical Association. He was a charter member and past-president of the Brooklyn Ophthalmological Society. He also was serving his second term as a member of the Grievance Committee of the New York State Department of Education.

Dr. Mallon was a zealous member of the Catholic Church and was very active in the founding of the Catholic Physicians Guild of Brooklyn.

Dr. Mallon died June 24, 1956, at Avon, New Jersey, at the age of 61. He is survived by his wife.

JOHN McGUIRE

Dr. John McGuire was born March 15, 1889, at Cedar Bluff, Virginia. He received his preliminary education at Emory and Henry College, Emory, Virginia, and his degree as Doctor of Medicine from the Medical College of Virginia. Postgraduate work was done at Manhattan Eye, Ear, Nose and Throat Hospital, New York City. Dr. McGuire began private practice in 1915 in Tazewell County, Virginia.

Dr. McGuire was a specialist certified by the American Board of Otolaryngology, and a member of the American Academy of Ophthalmology and Otolaryngology, the American Medical Association, West Virginia and Virginia Medical societies, Southern Medical Association, and the West Virginia Academy of Ophthalmology and Otolaryngology. He served on the staffs of St. Luke's Hospital, and Bluefield Sanitarium.

Hunting, fishing, and farming were Dr. McGuire's hobbies. He also was a member of the First Methodist Church; and he had served in World War I.

On June 9, 1956, Dr. McGuire died at Bluefield Sanitarium. Surviving are two sisters and one brother.

TOM OTTO WALTER MEISSNER

Dr. Tom Otto Walter Meissner, who was born in Vienna, Austria, August 3, 1911, died in an automobile accident near Athens, Georgia, on September 29, 1955.

Dr. Meissner received his degree in medicine from the Medizinische Fakultat der Universitat, Vienna, Austria, in 1936. He interned at the Passavant Hospital in Pittsburgh and did postgraduate work in ophthalmology at the Meller Clinic in Vienna, and at the Lawson Veterans Administration Hospital in Chamblee, Georgia.

Dr. Meissner began the practice of his specialty in Athens, Georgia, in 1950. He was affiliated with the Lawson Veterans

Administration Hospital, Athens General Hospital, St. Mary's Hospital, and the Emory University School of Medicine.

A diplomate of the American Board of Ophthalmology, Dr. Meissner was a Fellow of the American Academy of Ophthalmology and Otolaryngology, and a member of the American Medical Association and of state and local medical societies.

Dr. Meissner was a well-loved citizen of Athens and, during the few years of his practice there, had made a valuable contribution to the eye health of the community. His wife, Lola Burns Meissner, and one son, David, survive.

SAMUEL ARCHARD MORRIS

Dr. Samuel Archard Morris died in Los Angeles on December 3, 1955, at the age of 51 years.

Dr. Morris received his degree as Doctor of Medicine from McGill University in Montreal, Canada, in 1929. His postgraduate work was done at Bellevue Hospital, New York, New York Hospital and the Post-Graduate Hospital in New York. He began the practice of medicine in 1931, at Marysville, California. At the time of his death, he was affiliated with the Hollywood Presbyterian Hospital and the Cedars of Lebanon Hospital in Los Angeles.

Dr. Morris became a diplomate of the American Board of Ophthalmology in 1939, and was a Fellow of the American Academy of Ophthalmology and Otolaryngology and of the American College of Surgeons. He held membership in the American Medical Association and in the Los Angeles County Medical Association.

Dr. Morris is survived by his wife, Evelyn, and two sons.

WILFRID EDWARD MULDOON

Dr. Wilfrid Edward Muldoon was born in Youngstown, Ohio, on November 29, 1896. He was graduated by the University of Michigan in 1920, and received his degree as Doctor of Medicine from the University of Michigan Medical School in 1922. He did postgraduate work at the University of Paris, the University of Bordeaux and the Ancon Hospital in the Canal Zone. In 1923, he began the practice of medicine at the Hospital of the Tela Railroad in Honduras.

In 1926, Dr. Muldoon resumed his postgraduate studies at the University of Vienna. After a residency in ophthalmology at the Wills Eye Hospital in Philadelphia, he moved to San Antonio, Texas, where he engaged in the practice of ophthalmology. He was a consultant at Brooke General Army Hospital and in September 1950 was appointed associate professor of clinical ophthalmology on the faculty of Baylor University.

Dr. Muldoon, a diplomate of the American Board of Ophthalmology, was a Fellow of the American Academy of Ophthalmology and Otolaryngology. He was a member of the Association Medico Hondureno and of the American Medical Association of Vienna, and he held memberships in the American Medical Association and state and local medical societies.

Dr. Muldoon died on October 3, 1955, and is survived by his wife, Laurie; a son, William Henry; and a daughter, Laurie.

GEORGE WAVERLY NAIRN

Dr. George Waverly Nairn was born in Carroll, Nebraska, on February 6, 1895. He received his undergraduate education in Carroll and at Boulder, Colorado. He attended the University of Colorado School of Medicine, where he received his degree as Doctor of Medicine in 1922. In 1923, he began the practice of medicine in Douglas, Wyoming.

Dr. Nairn began his special training in otolaryngology and ophthalmology under the United States Veterans Bureau at the New York Post-Graduate Hospital and at the College of Physicians and Surgeons at Columbia University in 1925 and 1926. In September 1926 he established the Eye, Ear, Nose and Throat Department in the Wyoming County Community Hospital, Warsaw, New York; he was actively engaged on the staff of the hospital until his death.

Dr. Nairn was a diplomate of both the American Board of Otolaryngology and the American Board of Ophthalmology. He was a Fellow of the American Academy of Ophthalmology and Otolaryngology, and was a member of the Buffalo Ophthalmologic Club, of the American Medical Association, and of state and local medical societies.

Dr. Nairn died in Warsaw, New York, on October 28, 1955. He is survived by his wife, Ida, two daughters, and a son.

CHARLES FRANCIS NUTTER

Dr. Charles Francis Nutter was born on May 24, 1870, in Rochester, New Hampshire. He received his degree as Doctor of Medicine from the Medical School of Maine in Portland in 1922, and did postgraduate work at Maine General Hospital in Portland. He began the practice of medicine at Nashua, New Hampshire, in 1894.

During the First World War, Dr. Nutter served under Dr. John M. Wheeler and became interested in the specialty of ophthalmology. Upon his return to Nashua after the war, he limited his practice to that specialty. He was on the staff of Nashua Memorial Hospital and of St. Joseph's Hospital in Nashua.

Dr. Nutter was a Life Fellow of the American Academy of Ophthalmology and Otolaryngology and a fellow of the American College of Surgeons. He was also a member of the American Medical Association and of the state and local medical societies.

Dr. Nutter died in Nashua on December 22, 1955, at the age of 85 years. His sole surviver is his wife.

JOHN NELSON OSBURN

Dr. John Nelson Osburn was born in Rippon, West Virginia, on February 21, 1886. After graduating from Hampden-Sydney College in 1905, he represented the third generation in his family to study medicine at the University of Maryland School of Medicine, where he received his medical degree in 1909. He served as an intern in the University Hospital in Baltimore in 1909-1910, and later as a resident in the Episcopal Eye, Ear and Throat Hospital in Washington, D. C. Subsequently, he took postgraduate work in ophthalmology at Johns Hopkins Hospital, the Manhattan Eye, Ear and Throat Hospital, and the Knapp Clinic in New York.

Dr. Osburn practiced in Martinsburg, West Virginia, from 1910 to 1917. His practice was interrupted by the advent of World War I, in which he served as a Lieutenant in the Navy from 1917 to 1920. During this period, he was a Chief Medical Officer at the United States Naval Academy in Annapolis. After the war, he moved to Southern California and engaged in the practice of his specialty in Los Angeles until 1949, when he entered into semiretirement in the Harbor Area, where he had long been known as a summer resident. He carried on a limited consulting practice in Corona del Mar until forced into retirement by ill health in 1954.

Occupying a position of leadership in his specialty, Dr. Osburn's interests extended into various related fields. A pioneer in industrial accident work, he was recognized as an authority on the treatment of eye injuries. He was for many years chief of the Eye and Ear Department in the Santa Fe Coast Line Hospital in Los Angeles, and was the chief surgeon for the company in 1947-1948. Interested in ophthalmic aviation medicine, he did much of this important work for the air lines. In connection with sports, he served as one of the official physicians for the Olympic Games in Los Angeles in 1932, and he had been an advisor to the team physicians for the athletic department of the University of Southern California.

Dr. Osburn, a diplomate of the American Board of Ophthalmology, was a Fellow of the American Academy of Ophthalmology and Otolaryngology. He was a past president of the Ophthalmology-Otolaryngology and Industrial Medicine sections of the Los Angeles County Medical Association, and was also a member of the Los Angeles Ophthalmological Society, the Pacific Coast Oto-Ophthalmological Society, and the Aero Medical Association of the United States. He had served on the staffs of the Santa Fe, Good Samaritan, St. Vincent's, Children's, and Eye and Ear hospitals in Los Angeles, and of St. Joseph and Hoag Memorial-Presbyterian hospitals in Corona del Mar.

Dr. Osburn died at his home on Bay Island, in Orange County, California, on October 11, 1955. He is survived by his wife, Barbara Stephens Osburn, and two daughters.

ROBERT WRIGHT OWSLEY

Dr. Robert Owsley was born in Thorntown, Indiana, on January 10, 1912. His undergraduate education was obtained at Indiana University. He attended the Indiana University School of Medicine in Indianapolis, where he received his degree as Doctor of Medicine in 1934. The following year, he began the practice of medicine as a general practitioner in his native city.

In 1938, Dr. Owsley engaged in postgraduate work in otolaryngology at Washington University School of Medicine in St. Louis. He became a diplomate of the American Board of Otolaryngology in 1941. During the following years, 1942 through 1946, he served in the Medical Corps of the Air Force, being stationed at Randolph Field, San Antonio Aviation Medical Center, School of Air Evacuation at Bowman Field, Langley Field, Westover Field, and Mitchell Field. He retired in 1946 with the rank of Lieutenant Colonel.

At the time of his death in San Francisco on August 15, 1955, Dr. Owsley was engaged in the practice of otolaryngology in Arcadia, California.

Dr. Owsley was a Fellow of the American Academy of Ophthalmology and Otolaryngology and a member of the Pacific Coast Oto-Ophthalmological Society, the American Medical Association, and state and local medical societies.

Dr. Owsley is survived by his wife, Helen Owsley; a son, and two daughters.

JAMES DEVOTE PERDUE

Dr. James Devote Perdue was born in Farmersville, Alabama, on January 30, 1889. He received his degree of Doctor of Medicine from the University of Alabama in 1913 and began the practice of medicine in Mobile, Alabama, in the same year. After completing postgraduate work in ophthalmology at Tulane University in New Orleans, and at Professor Doctor Heinrich Neumann's Clinic in Vienna, he became a diplomate of the American Board of Ophthalmology in 1927. He was on the retired staff of the Mobile Infirmary, of Providence Hospital, and of City Hospital in Mobile. For many years, he was a member of the Board of Censors of the Alabama State Medical Association.

Dr. Perdue was a Fellow of the American Academy of Ophthalmology and Otolaryngology and of the American College of Surgeons. He was a member of the American Medical Association and of state and local medical societies.

Dr. Perdue died in Mobile on May 18, 1956. He is survived by his wife, Ayrault Chapman Perdue.

JAMES ROSS REED

Dr. James Ross Reed died in Pasadena, California, on May 11, 1955.

Dr. Reed was born in Erie, Pennsylvania, on February 24, 1880. He received his undergraduate education at the University of Michigan, and was granted his degree as Doctor of Medicine from the University of Michigan Department of Medicine and Surgery in 1905. After an internship at Carney Hospital in Boston, he did postgraduate work in ophthalmology and otolaryngology at the New York Eye and Ear Infirmary. He began the practice of his specialty in Pasadena, California, in 1908.

During Dr. Reed's active practice in California, he was affiliated with the University of Southern California School of Medicine and was attending ophthalmologist at Los Angeles General Hospital. He served as consulting ophthalmologist and chief of staff at Huntington Memorial Hospital in Pasadena and held the position of senior ophthalmologist at St. Luke Hospital in Pasadena.

Dr. Reed was a diplomate both of the American Board of Ophthalmology and of the American Board of Otolaryngology. He was a Fellow of the American Academy of Ophthalmology and Otolaryngology, of the American College of Surgeons, and of the International College of Surgeons. He held membership in the Pacific Coast Oto-Ophthalmological Society and the Los Angeles Society of Ophthalmology and Otolaryngology.

Surviving Dr. Reed are his wife, Margaret, and five sons.

CLARENCE EDWARD ROBBINS

Dr. Clarence Edward Robbins was born December 1, 1899. He graduated in 1923 from Harvard Medical School, receiving his degree of Doctor of Medicine. He had received his Bachelor of Arts degree from the University of North Dakota.

In 1925 Dr. Robbins was certified a specialist by the American Board of Otolaryngology. He was on the staff of St. Mary's Hospital, Pierre, South Dakota, and was a member of the South Dakota State Medical Association, the American Medical Association, and the South Dakota Academy of Ophthalmology and Otolaryngology. He was a Fellow of the American Academy of Ophthalmology and Otolaryngology and of the American College of Surgeons.

Dr. Robbins was associated with the Pierre Clinic at Pierre, South Dakota, in

1950, when he suffered a severe stroke. He died May 27, 1956. Surviving is his widow, Mrs. C. E. Robbins.

WILLIAM HUMES ROBERTS

Dr. William Humes Roberts died September 18, 1955, at LaJolla, California. He was a specialist certified by both the American Board of Ophthalmology and the American Board of Otolaryngology. Dr. Roberts was a Life Fellow of the American Academy of Ophthalmology and Otolaryngology. He also held membership in the American Laryngological, Rhinological and Otological Society; he was a fellow of the American College of Surgeons and was an associate member of the American Medical Association.

Dr. Humes practiced at Pasadena, California, until July of 1945, when he retired.

Surviving is his wife, Mrs. William H. Roberts of La Jolla, California.

HENRY PAUL SCHUGT

Dr. Henry Paul Schugt was born June 19, 1895. In 1920 he graduated from Julius-Maximilians-Universitat Medizinische Fakultat, Wurzburg, Bavaria, Germany.

Dr. Schugt was associate otolaryngologist at Lenox Hill Hospital and at Lutheran Hospital, both in New York City. He was chief otolaryngologist at Lenox Hill Hospital Dispensary, and he was consultant at St. Francis Hospital in Port Jervis, New York. He was a Fellow of the American Academy of Ophthalmology and Otolaryngology and of the American College of Surgeons.

Dr. Schugt died January 30, 1956, at Lenox Hill Hospital at the age of 60, of cerebral hemorrhage.

JAMES WASHINGTON SMITH

Dr. James Washington Smith was born in Washington, D. C., on May 16, 1893. He was educated in the public schools of Washington and New York and received his degree as Doctor of Medicine at New York University, University and Bellevue Hospital Medical College in New York City in 1917. He did his postgraduate work in ophthalmology at the hospital of his alma mater. He began the practice of his specialty in New York City in 1919.

Dr. Smith served as clinical professor of ophthalmology at the Post-Graduate Medical School of the New York University. He was also chief of the eye service at the Hospital for Joint Diseases, consultant ophthalmologist at Goldwater Hospital, attending ophthalmologist at University Hospital, a member of the Board of Visitors at Rockland State Hospital, and a member of the courtesy staff of the Manhattan Eye and Ear Hospital.

Dr. Smith was the author of many contributions to medical literature. He was known as the inventor of the blepharitis comb. In 1935, he received the New York University Alumni Meritorious Award.

A diplomate of the American Board of Ophthalmology, Dr. Smith was also a Fellow of the American Academy of Ophthalmology and Otolaryngology and of the American College of Surgeons. In 1951-52, he served as chairman of the New York Academy of Medicine, Section on Ophthalmology. He held membership in the Association for Research in Ophthalmology and was a past president and secretary of the New York University College of Medicine Alumni.

Dr. Smith was interested in Americana with especial interest in Presidential mail. In his collection, he had letters from all of the Presidents, and his library held the first editions of many rare books. He was a charter member of the Manuscript Society.

Dr. Smith died in New York on October 24, 1955. He is survived by his wife, Sara, and a son, Dr. Barry F. Smith.

WILLIAM HAMILTON SMITH

Dr. William Hamilton Smith was born March 25, 1883. In 1906 he graduated from George Washington University School of Medicine, Washington, D. C., with the degree of Doctor of Medicine. About 1912 he became interested in otolaryngology, and he was practicing this specialty exclusively in 1915.

Dr. Smith was a consultant on the staff of Washington County Hospital at Hagerstown, Maryland. He was a Fellow of the American Academy of Ophthalmology and Otolaryngology and held membership in the Cumberland Valley Medical Association; American Medical Association; Medical and Chirurgical Faculty of the State of Maryland; and

Washington County Medical Society. He was also a fellow of the American College of Surgeons. He had served in World War I.

Dr. Smith died November 7, 1955, at the age of 72.

CARL BROSIUS SPUTH

Dr. Carl Brosius Sputh was born in Indianapolis, Indiana, on October 4, 1884. He received a degree of Bachelor of Physical Education from the Normal College of the American Gymnastic Union (now a part of Indiana University) in 1904, and his degree as Doctor of Medicine from Indiana University School of Medicine, in Indianapolis, in 1913. In the same year, he began the practice of medicine in Indianapolis. Postgraduate work in ophthalmology and otolaryngology was done at Chicago Eye, Ear, Nose and Throat Hospital, Harvard University, Indiana University and Johns Hopkins University.

Dr. Sputh was associate professor of ophthalmology at the Indiana University School of Medicine and served as chief ophthalmologist and otolaryngologist for the United States Employees Compensation Commission in Wisconsin. He was affiliated with the Indiana General Hospital and was a member of the consulting staffs of the Methodist and St. Vincent's hospitals in Indianapolis, and of St. Francis Hospital in Beech Grove, Indiana.

A diplomate of the American Board of Otolaryngology, Dr. Sputh was a Fellow of the American Academy of Ophthalmology and Otolaryngology and of the International College of Surgeons. He held membership in the Indiana Academy of Ophthalmology and Otolaryngology, in the American Medical Association, and in state and local medical societies.

Dr. Sputh was always interested in physical education. In 1910, he had organized and directed the first playground in Indianapolis. From 1912 to 1953, he served as president of the Athenaeum Turners. From 1913 to 1916, he organized and directed the School of Physical Education at State Teachers College, La Crosse, Wisconsin. In 1934, he was appointed president of the Normal College of the American Gymnastic Union, where he served until his retirement in 1941.

Dr. Sputh died in his native city on April 8, 1956. He is survived by his wife, Elsa; a daughter, Mrs. John Hash; and a son, Dr. Carl B. Sputh, Jr., who is also a member of the Academy.

HERMON MARSHALL TAYLOR

Dr. Hermon Marshall Taylor was born in Wilmington, North Carolina, on June 7, 1881. He spent his early life in Lexington, Virginia, where he lived in the old home of Stonewall Jackson. He received his medical education at Emory University in Atlanta and was awarded the degree of Doctor of Medicine by that institution in 1903. Later he engaged in postgraduate study at the Medical College of Virginia in Richmond. Training in his specialty was obtained at the New York Eye and Ear Infirmary. He studied otolaryngology in the clinics of Vienna, Munich and Paris. During World War I, he served as a captain in the Medical Corps of the United States Army.

In 1911, Dr. Taylor located in Jacksonville, Florida, where for more than four decades he engaged in the practice of his specialty. He served as consulting otolaryngologist to the Duval Medical Center, to Riverside Hospital, and to St. Luke's Hospital, where he was also chief of staff. He was a member of the staffs of St. Vincent's, Hope Haven, and Brewster hospitals. In addition, for many years he was consulting otolaryngologist to the Florida East Coast Hospital and the State School for the Deaf and Blind at St. Augustine.

Dr. Taylor was a Fellow of the American Academy of Ophthalmology and Otolaryngology. A former president of the Florida Medical Association and of the Duval County Medical Society, for a number of years he represented Florida in the House of Delegates of the American Medical Association. He was a founding member and first president of the Florida Society of Ophthalmology and Otolaryngology. He served as head of the Department of Otolaryngology of the Graduate School of Medicine of the University of Florida since its founding and was the founder of its Midwinter Seminar in Ophthalmology and Otolaryngology.

Dr. Taylor served terms as secretary and as chairman of the Section on Ophthalmology and Otolaryngology of the Southern Medical Association. He was on the Council of the Association for six

years, serving as its chairman for the last two years, and then became president of the Association in 1935.

In addition to serving as a member of the House of Delegates of the American Medical Association for some years, Dr. Taylor in 1938 was chairman of the Section on Laryngology, Otology and Rhinology of that organization. For twelve years he held the chairmanship of its Committee on Otorhinologic Hygiene of Swimming.

Dr. Taylor was a fellow of the American College of Surgeons, and in 1950 he was elected to its Board of Governors. In 1935, he was president of the American Bronchoscopic Society, now known as the American Broncho-esophagological Association. He acceded to the presidency of the American Laryngological, Rhinological and Otological Association in 1944. He became president of the American Laryngological Association in 1952. This society bestowed upon him its highest honors for outstanding achievements—the Casselberry Award in 1939 and the James E. Newcomb Award in 1950.

Dr. Taylor found time in his busy practice to engage in fruitful clinical research and to contribute many articles which have enriched the literature of his specialty.

While participating on September 11, 1955, in the opening ceremonies of the Baptist Hospital of Jacksonville, for which he had labored arduously, serving in many official capacities, Dr. Taylor was stricken with coronary occlusion and cerebral thrombosis. He died on September 22, 1955. Dr. Taylor is survived by his wife, Pallie Elaine Dekle Taylor, and three children: Margaret, who is Mrs. W. J. Pattison, and two sons, Coakley Taylor, a Jacksonville attorney, and Dr. G. Dekle Taylor, who is also a Fellow of the Academy.

PETER HUNTER THOMPSON

Dr. Peter Hunter Thompson, a Life Fellow of the American Academy of Ophthalmology and Otolaryngology, died in Boston, Massachusetts, on December 1, 1955.

Dr. Thompson was a native of Canada, having been born at Brigham, Quebec, on May 3, 1873. He was the youngest of 12 children, orphaned by his father's death when he was two years old. His professional education and training were secured entirely through his own efforts. He received a degree of Doctor of Medicine from Tufts College Medical School in Boston in 1898, and from Harvard Medical School in 1901. He was formerly on the faculty at Harvard Medical School in the Department of Ophthalmology and was affiliated with Boston City Hospital and the Massachusetts Eye and Ear Infirmary. He was a member of the consulting staff of the Massachusetts General Hospital.

Dr. Thompson was a diplomate of the American Board of Ophthalmology. He was past president of the New England Ophthalmological Society, and he was a member of the American Medical Association and of state and local medical societies.

Dr. Thompson was well known for his ability as a diagnostician and for his meticulous refractions. He was dedicated to his profession and his many patients held him in high regard. Dr. Thompson is survived by one son, Peter Hunter Thompson, Jr.

ROBERT RUSSELL TRACHT

Dr. Robert Russell Tracht was born December 5, 1899. In 1932 he graduated from Loyola University School of Medicine, Chicago, with the degree of Doctor of Medicine.

In 1937 Dr. Tracht began to practice ophthalmology exclusively. He was a specialist certified by the American Board of Ophthalmology. He served on the faculty of the University of Minnesota Medical School, Minneapolis, and of the University of Minnesota Graduate School through his association with the Mayo Foundation of Rochester, Minnesota. He was on the staff of the Charles T. Miller Hospital and was refractionist at the Wilder Clinic, St. Paul, Minnesota. Dr. Tracht was a Fellow of the American Academy of Ophthalmology and Otolaryngology, and he held membership in the American Ophthalmological Society, the Minnesota State Medical Society, Ramsey County Medical Society, the American Medical Association, and the Association for Research in Ophthalmology.

Dr. Tracht died in St. Paul on May 25, 1956, at the age of 56.

IVAN FORD WEIDLEIN

Dr. Ivan Ford Weidlein was born in 1898 at Wellman, Iowa. His premedical

and medical education was obtained at the University of Iowa, where he received his medical degree in 1922.

After serving an internship at University Hospitals, Iowa City, Iowa, from 1922 to 1924, Dr. Weidlein spent two years as resident in otolaryngology at Lakeside Hospital, Cleveland. He was certified by the American Board of Otolaryngology in 1928. He studied his specialty in Vienna for six months in 1929.

Dr. Weidlein began his practice of otolaryngology in Cleveland in July 1926 and continued in that city until his death on June 17, 1955.

From 1927 to 1944 Dr. Weidlein served on the teaching staff of the University Hospitals, Cleveland, advancing from clinical instructor to assistant professor in otolaryngology at Western Reserve University Medical School. From 1944 until his death he was otolaryngologist at Doctors Hospital, Cleveland Heights.

A Senior Fellow of the American Academy of Ophthalmology and Otolaryngology, Dr. Weidlein was also a member of the American Medical Association and of the Cleveland Otolaryngological Club.

Aside from his profession, Dr. Weidlein maintained an interest in farming. He was a member of the Presbyterian church.

Surviving Dr. Weidlein are his wife, Eleanor Bartal Weidlein, a son and a daughter.

SAMUEL BROWNELL WESTLAKE

Dr. Samuel Brownell Westlake, a Life Fellow of the American Academy of Ophthalmology and Otolaryngology, died on March 27, 1956, in St. Louis, Missouri, at the age of 77 years.

Born in Brooklyn on February 9, 1879, Dr. Westlake spent his early life in the eastern part of the United States. He attended Mt. Herman Boy's School in Northhampton, Massachusetts, and Norwich Academy in Norwich, Connecticut. Dr. Westlake attended Norwich Medical College and there received the degree of Doctor of Medicine in 1906. Subsequently, he obtained postgraduate training at St. Louis University School of Medicine and at the Polyklinik, University of Vienna. He was certified by the American Board of Otolaryngology in 1925.

Dr. Westlake began the practice of medicine in Baltimore, Maryland, in 1906. After moving to St. Louis, he became associated with the hospitals connected with the St. Mary's Group of Hospitals of St. Louis University School of Medicine and was assistant in otolaryngology at the school from 1913 to 1916, later serving as instructor and assistant professor. At Jewish Hospital, St. Louis, he became assistant laryngologist in 1913, associate laryngologist in 1925 and director of the Department of Otolaryngology in 1934, serving until 1948. He was otolaryngologist-in-chief of the Missouri Pacific Hospital, St. Louis, from 1928 to 1948.

Among the medical organizations of which Dr. Westlake was a member are St. Louis Medical Society, Missouri State Medical Society, American Medical Association, American Laryngological, Rhinological and Otological Society, and the American Otological Society, as well as the American Academy of Ophthalmology and Otolaryngology.

Dr. Westlake was a member of the Presbyterian church. Yachting, hunting and horseback riding were among his favorite activities.

During World War I, Dr. Westlake served as a captain in the Medical Corps, United States Army. He was chief of the otological service at Camp Sherman, in 1917.

Dr. Westlake is survived by his wife; two sons, Samuel B., Jr., and William W.; a daughter, Martha (Mrs. Curtis Reeves), and five grandchildren.

ANTHONY CHARLES ZEHNDER

Dr. Anthony Charles Zehnder was born in Newark, New Jersey, on February 6, 1881. He died at his home in Newark on December 1, 1955.

Dr. Zehnder's early education was obtained at St. Mary's School and St. Benedict's Academy, Newark. He entered Cornell Medical College in 1903 and received his medical degree in 1907. He interned at Bellevue Hospital, New York City, from 1907 to 1909, serving as junior, senior, and house surgeon on the Second (Cornell) Surgical Division.

The first summer after internship Dr. Zehnder spent as camp physician at the Adirondack League Club, and he often used to tell of his experiences and the pleasures he had in his first opportunity

as a physician. He opened his office in Newark in 1910 and became associated with St. James, St. Michael's, and Newark City hospitals, and the Newark Eye and Ear Infirmary. These years were spent as a general practitioner, but his interest in his future specialty, ophthalmology, began at the Newark Eye and Ear Infirmary, where he was associated with Dr. Charles J. Kipp, Dr. Wells P. Eagleton, and Dr. Charles F. Webner. These men were to become his preceptors in ophthalmology.

The day after World War I was declared in 1917, Dr. Zehnder volunteered for service and was commissioned a first lieutenant in the Army of the United States. He was sent to Princeton, New Jersey, and became post surgeon at the Flight Training School at Princeton University. From Princeton he was ordered to Telliafor Field, Texas, and became one of the first flight surgeons. This department in 1917 was a part of the Signal Corps. He was discharged in 1918 from the Army with the rank of Captain. In those days flying was a new adventure, and he often related some of the experiences he had in flying to crashes and accidents. They all made a lasting impression, for it was many years before he would ever think of flying again. Only in recent times did he fly, and then with his children who had married and moved out of Newark.

Dr. Zehnder resumed his medical practice in 1918, this time establishing himself as a specialist in ophthalmology. He became attending ophthalmologist at the Newark Eye and Ear Infirmary and the Presbyterian Hospital of Newark, and consultant ophthalmologist at Mountainside Hospital, Montclair.

In 1919, Dr. Zehnder was elected a Fellow of the American Academy of Ophthalmology and Otolaryngology. Having been a member for fifty years in 1949, he became a Life Fellow of the organization. Dr. Zehnder was a member of the American Medical Association, the Academy of Medicine of New Jersey, the Essex County Medical Society, the Society of Surgeons of New Jersey, the Society of Alumni of Bellevue Hospital, and the Practitioners and the Doctors clubs. He was elected president of the Essex County Society of New Jersey in 1935 and served for one term.

Members of Dr. Zehnder's family who survive him are a daughter, Mrs. Jack Camblos of Charlottesville, Virginia; two sons, William of Greenwich, Rhode Island, and Charles, who is attending school in Providence, Rhode Island; and a sister, Miss Rena Zehnder of East Orange, New Jersey.

News - - - Notes

SECTION ON OPHTHALMOLOGY A.M.A.

Dr. T. E. Sanders and Dr. Albert P. Ley were awarded a $250.00 prize for their paper "Fungus Keratitis" at the meeting of the Section on Ophthalmology, American Medical Association, in Chicago in June 1956. The award was made on on the basis of presentation and originality.

SECTION ON OPHTHALMOLOGY AND ASSOCIATION FOR RESEARCH IN OPHTHALMOLOGY

CALL FOR PAPERS

The 1957 meeting of the Section on Ophthalmology will be held in New York, June 3-7, 1957, simultaneously with that of the Association for Research in Ophthalmology.

Anyone wishing to present a paper before the Section is urged to communicate with Dr. Harold G. Scheie as soon as possible. A title and abstract of 50 to 150 words must be in his hands by Jan. 1, 1957. Anyone wishing to present a paper before the Association for Research should contact Dr. Lorand V. Johnson as soon as possible.

Harold G. Scheie, Secretary
Section on Ophthalmology
313 South 17th St.
Philadelphia 3, Pa.

Lorand V. Johnson, Secretary
Association for Research in Ophthalmology
10515 Carnegie Ave.
Cleveland 6, Ohio

CALIFORNIA

Urgent Request

The Uveitis Laboratory, University of California School of Medicine, San Francisco, is anxious to obtain freshly enucleated eyes from patients with **all types of uveitis** and endogenous uveitis. We are making attempts to isolate viruses and other etiologic agents from these eyes. We are also interested in pathologic studies to aid in identification of any agents. In addition to eyes with the usual types of uveitis, we are interested in those which develop intraocular inflammations as a result of sclerosing keratitis, herpes febrilis and zoster of the cornea, mycotic corneal infections, glaucoma secondary to endogenous uveitis, Coats' disease, Eales' disease, papillitis and scleritis. The eye tissues will be cultured in various media.

The eyes should **not** be fixed in preservatives or frozen, but placed in a sterile bottle, packaged, and shipped as quickly as possible. Please send specimens air express, special delivery, **collect.** Enclose history and findings and mark the package "Fresh Tissue Specimen—Rush."

A report of isolations of organisms and pathologic findings, including a slide, will be sent to the contributor, and credit will be given in any resulting publications if desired.

Please telegraph collect if a specimen is being sent. Send eyes to Samuel J. Kimura, M.D., Michael J. Hogan, M.D., or Phillips Thygeson, M.D., University of California School of Medicine, San Francisco 22, California.

The Section on Eye, San Francisco Medical Society, will meet at 8:15 p.m., December 4, 1956, at 250 Masonic Ave., San Francisco. The following program will be presented:

1. Consideration of Professional Liability (Malpractice Insurance)
2. Case Presentations, by residents of University of California and Stanford University Medical School departments of ophthalmology
3. Election of Officers

FLORIDA

The Eleventh Annual University of Florida Midwinter Seminar in Ophthalmology and Otolaryngology will be held January 14-19, 1957. As in previous years, Miami Beach will be the host city.

The lectures on Ophthalmology will be presented on January 14, 15, and 16. The lecturers for the courses in Ophthalmology will be Irving H. Leopold, M.D., Philadelphia; Cecil W. Lepard, M.D., Detroit; Francis Heed Adler, M.D., Phila-

delphia; Algernon B. Reese, M.D., New York City; and Harold W. Brown, M.D., New York City.

Outstanding teachers who will lecture on January 17, 18, and 19 are Alden H. Miller, M.D., Los Angeles; Porter P. Vinson, M.D., Richmond, Virginia; Lester Hale, M.D., Gainesville, Florida; James B. Costen, M.D., St. Louis, and Philip Meltzer, M.D., Boston.

ILLINOIS

Dr. William M. S. Ironside of The Royal Infirmary, Edinburgh, Scotland, has been appointed assistant professor of otolaryngology in the Department of Surgery of the University of Chicago, according to announcement by Dr. Lowell T. Coggeshall, dean of the Division of Biological Sciences of the University of Chicago. The appointment is effective this Fall.

This will be Dr. Ironside's second period of service at the University of Chicago. He was a fellow and instructor in otolaryngology at the University for the year ending July 31, 1955.

Dr. Ironside obtained the degree of Doctor of Medicine at the University of Aberdeen, Scotland, in 1945. He became a fellow the Royal College of Surgeons, Edinburgh, in 1953. He was born in Invergordon, Rosshire, Scotland.

Mrs. Ironside is a physician also, and they have a daughter three and one-half years old.

LOUISIANA

The Mid-Winter Convention of the New Orleans Academy of Ophthalmology will take place February 11-15, 1957. The program will feature a Symposium on Glaucoma by Bernard Becker, M.D., W. Morton Grant, M.D., Joseph S. Haas, M.D., A. E. Maumenee, M.D., Harold G. Scheie, M.D., Kenneth C. Swan, M.D., and Georgiana D. Theobald, M.D.

The registration fee of $75.00 includes associate membership in the New Orleans Academy of Ophthalmology for the year 1957, as well as all other features of the convention.

MASSACHUSETTS

Dr. Gerald Fonda and Dr. Charles Schepens will join the AO Bureau of visual Science staff for the third in American Optical Company's series of conferences on Visual Optics and Refraction for ophthalmologists. The meetings will be in Southbridge Nov. 12, 13 and 14.

Dr. Fonda, of New York, will lecture on "Correction of Subnormal Vision," and Dr. Schepens, of Boston, on "Indirect Ophthalmoscopy."

Subjects to be covered by Bureau of Visual Science staff members include "Basic Concepts in Ophthalmic Optics," "Objective Refractive Tests," "Physiological Methods of Determining Multifocal Corrections," "Ophthalmic Lens Characteristics," "The H-R-R Color Vision Test," "Monoplex Eyes and Implants," and "Clinical Aspects of Aniseikonia."

These courses are open to all ophthalmologists, and are particularly helpful to physicians preparing to enter the field of ophthalmology. There is no charge for participation in the course. Advance registration is advisable since attendance will be limited. Full details may be obtained from the Bureau of Visual Science at American Optical Company, Southbridge, Mass.

MINNESOTA

A testimonial dinner was held at the Town and Country Club, St. Paul, Minnesota, on September nineteenth, honoring Dr. Erling W. Hansen, professor of ophthalmology at the University of Minnesota Medical School, celebrating his election as President of the American Academy of Ophthalmology and Otolaryngology. Eighty-two of his friends and colleagues attended, including thirty-two of Dr. Hansen's former residents and students. A gold-banded gavel inscribed with an announcement of the occasion and a gold wrist-watch band were presented to Dr. Hansen by his former associates and students.

NEW YORK

Surgery of the Cornea will be offered as a full-time course by the New York University Post-Graduate Medical School, December 3-7, 1956.

Given under the direction of Dr. Ramon Castroviejo, the course is designed to cover thoroughly the current concepts and practices in the field of corneal surgery by lectures, observations of surgical procedures in the operating room, and practice on animal eyes.

Surgery of the Cornea is one of eleven courses offered by the Post-Graduate Department of Ophthalmology during the academic year. For further information write to the Dean, Post-Graduate Medical School, 550 First Ave., New York 16, N. Y.

PENNSYLVANIA

Temple University will offer the following postgraduate courses in 1957:

Postgraduate Course in Bronchoesophagology

February 11-22
May 20-31
September 9-20

Postgraduate Course in Laryngology and Laryngeal Surgery

March 11-22
November 4-15

These courses are all to be given in the Department of Laryngology and Bronchoesophagology, Temple University Hospital and School of Medicine, under the direction of Doctors Chevalier L. Jackson and Charles M. Norris.

The tuition fee for each course is $250.00.

Further information and application blanks can be obtained from Dr. Chevalier L. Jackson, 3401 N. Broad Street, Philadelphia 40.

ENGLAND

The Annual Congress of the Ophthalmological Society of the United Kingdom will be held at the Royal Society of Medicine, 1 Wimpole Street, London, W.1, on April 11, 12 and 13, 1957.

The Presidential Address will be delivered by Mr. J. J. Healy on "The Intraocular Circulation in Arterio-sclerosis and High Blood Pressure."

The subject for discussion will be "Ocular Aspects of Diabetes." This will be opened by Professor G. I. Scott, Dr. Norman Ashton and Dr. J. D. N. Nabarro. Members who wish to take part in the subsequent discussion are advised to intimate their intention before the opening of the Congress. It is emphasized that no member may speak for more than ten minutes.

There will also be a short symposium on "Congenital Cataract."

On this occasion the Bowman Lecture will be delivered by Sir Stewart Duke-Elder, his subject being "The Aetiology of Glaucoma."

Members wishing to read a short paper or to give a short pictorial demonstration are asked to send the title, together with an abstract, to Mr. J. R. Hudson not later than November 30, 1956. Those wishing to show films should also communicate with Mr. Hudson by the same date.

The Annual Dinner will be held at the Trocadero Restaurant on Thursday, April 11. Members may bring guests.

A Trade Exhibition will be held in the Cowdray Hall (next door to the Royal Society of Medicine).

On account of the difficulty in obtaining hotel accommodation in London, all members who will require it are advised to make their arrangements in good time.

The honorary secretaries of the Society are C. A. Cook (Council Business) and J. R. Hudson (Congress Business), 45 Lincoln's Inn Fields, London, W.C.2.

PRELIMINARY PROGRAM

SUNDAY, MAY 5

8:30 a.m.-4:00 p.m.—Registration, Statler Hotel
Scientific and Technical Exhibits open

5:00 p.m.—Convocation, Constitution Hall

6:00 p.m.—President's Reception, Pan American Union

MONDAY, MAY 6

8:30 a.m.-5:00 p.m.—Registration and Exhibits
(open daily during these hours)

9:00 a.m.-12:00 noon—Plenary Session:

CHRONIC SUPPURATION OF THE TEMPORAL BONE

Openers:

MARCUS DIAMANT, Sweden
Anatomical Etiologic Factors in Chronic Middle Ear Discharge

LUZIUS RUEDI, Switzerland
Pathogenesis and Treatment of Cholesteatoma in Chronic Suppuration of the Temporal Bone

HORST WULLSTEIN, Germany
Surgical Repair for Improvement of Hearing in Chronic Otitis Media

Discussers:

A. TUMARKIN, England
JUAN MANUEL TATO, Argentina
T. E. CAWTHORNE, England
FRITZ ZOLLNER, Germany

2:00-5:00 p.m.—General Sessions, 4 simultaneous Sections

2:00 p.m.—Tour, National Institutes of Health, Bethesda, Md.
Tour, Audiology and Speech Center, Walter Reed Army Medical Center (available daily)

3:30 p.m.—Tour, National Naval Medical Center, Bethesda, Md.
Tour, Audiology and Speech Center, Walter Reed Army Medical Center (available daily)

8:00 p.m.—Reception and Tour, National Gallery of Art

TUESDAY, MAY 7

9:00 a.m.-12:00 noon—General Sessions, 4 simultaneous Sections
Closed Circuit, Color Medical Television
Special all-day program for ladies

2:00-5:00 p.m.—General Sessions, 4 simultaneous Sections
Film Session

8:30 p.m.—Concert, Constitution Hall

WEDNESDAY, MAY 8

9:00-9:30 a.m.—Special address:

DOCTORS AS DIPLOMATS

HOWARD RUSK, Director, Institute of Physical Medicine and Rehabilitation, New York University-Bellevue Medical Center

9:30 a.m.-12:30 p.m.—Plenary Session:

(Continued on Next Page)

PRELIMINARY PROGRAM — *Continued*

COLLAGEN DISORDERS OF THE RESPIRATORY TRACT

Openers:
HANS SELYE, Canada
Introduction
MICHELE ARSLAN, Italy
The Upper Respiratory Tract
LESLIE GAY, U. S. A.
Experience with Steroids in the Treatment of Bronchial Asthma

Discussers:
VICTOR NEGUS, England
BRANIMIR GUSIC, Yugoslavia
AUBREY G. RAWLINS, U. S. A.
HENRY L. WILLIAMS, U. S. A.

2:00-5:00 p.m.—General Sessions, 4 simultaneous Sections
Film Session

5:00 p.m.—Business meeting, International Committee

7:00 p.m.—Dinner for official delegates and wives

THURSDAY, MAY 9

9:00 a.m.-12:00 noon—General Sessions, 4 simultaneous Sections
Closed Circuit, Color Medical Television

2:00-5:00 p.m.—General Sessions, 4 simultaneous Sections
Film Session

8:00 p.m.—Grand Banquet

FRIDAY, MAY 10

9:00 a.m.-12:00 noon—Plenary Session:

PAPILLOMA OF THE LARYNX

Openers:
Jo ONO, Japan
Etiology
PLINIO DE MATTOS BARRETTO, Brazil
Diagnosis
F. C. W. CAPPS, England
Therapy

Discussers:
C. A. HAMBERGER, Sweden
PEDRO HERNANDEZ GONZALO, Cuba
EELCO HUIZINGA, Netherlands
ALBERT VON RICCABONA, Austria

2:00-5:00 p.m.—General Sessions, 4 simultaneous Sections
Film Session

The following other international meetings will take place after the close of the International Congress of Otolaryngology:

Sixth International Congress of Bronchoesophagology, Philadelphia, Pa., May 12 and 13

International Conference on Audiology, St. Louis, Mo., May 14, 15, 16

International Voice Conference, Chicago, Ill., May 20, 21, 22

The American Express Company has arranged the following conducted tours to begin immediately after the International Congress of Otolaryngology:

TOUR 1: 7 days; will include Baltimore, Philadelphia, Boston, New York.

TOUR 2: 17 days; will visit Baltimore, Philadelphia, St. Louis, Iowa City, Rochester (Minn.), Chicago, Detroit, Niagara Falls, Boston, New York.

TOUR 3: 21 days; includes Baltimore, Philadelphia, St. Louis, Grand Canyon, Los Angeles, San Francisco, Chicago, Niagara Falls, New York.

TOUR A: 3 days; through Colonial Virginia visiting Charlottesville, Richmond, Williamsburg, Yorktown, Fredericksburg.

TOURS 1-A, 2-A, 3-A. First 3 days itinerary of Tour A. Tour 1-A will join Tour 1 in Boston; Tours 2-A and 3-A will proceed to St. Louis and from there complete the scheduled itinerary of Tour 2 or Tour 3 as the case may be.

GENERAL SECRETARY, 700 NORTH MICHIGAN AVENUE, CHICAGO 11, ILLINOIS, U.S.A.

POSITIONS AVAILABLE

Residency in Ophthalmology for term ending June 30, 1959; immediate vavancy. To be considered, applicant must be a U. S. citizen, graduate of approved American medical school or diplomate of National Board of Medical Examiners, and have completed an approved internship. Active 1300-bed general hospital with university affiliations. For information and forms of application, write Chief, Ophthalmology Section, Veterans Administration Hospital, Bronx 68, New York.

Ophthalmology Residency. As a result of the sudden unexpected loss of the senior resident, there is immediately available a senior assistant residency, or residency for a properly qualified man, on the medical school ophthalmology service. Very active participation in teaching and organized research, as well as busy clinic and surgical experience, is offered. Write Winston Roberts, M.D., Chief of Ophthalmology, Bowman Gray School of Medicine and North Carolina Baptist Hospital, Winston-Salem, North Carolina.

Ophthalmologist and Otolaryngologist needed in town of 35,000. Because of recent death of physician, an established practice and equipped offices are immediately available. Write Mrs. George H. Traugh, 722 Coleman Ave., Fairmont, West Virginia.

Senior Resident in Ophthalmology wanted for busy teaching service. Applicant must have had two or more years of residency training. This residency is for January 1, 1957 to July 1, 1957. Resident will be in charge of service with five other residents and will be responsible for between two hundred and three hundred major eye operative procedures. Interested qualified persons will please write to Richard G. Troutman, M.D., State University of New York, College of Medicine, 451 Clarkson Ave., Brooklyn 3, N. Y.

Ophthalmologist wanted by 14-man specialty group. Desire to establish Department of Ophthalmology. Group has own 65-bed air-conditioned hospital. Candidate must be certified or eligible. Salary open. Located in rapidly growing university city. Write Carbondale Clinic, Carbondale, Ill.

Otolaryngologist wanted by 14-man specialty group. Desire to establish Department of Otolaryngology. Group has own 65-bed air-conditioned hospital. Candidate must be certified or eligible. Salary open. Located in rapidly growing university city. Write Carbondale Clinic, Carbondale, Ill.

Ophthalmologist wanted to associate with part-time university faculty member in partially built-up practice. Immediate opportunity. Florida license required. Board certified or eligible. Percentage immediately, leading to partnership or purchase. University appointment available. Contact John F. McKenna, M.D,. 6915 Red Road, South Miami 43, Florida.

An opening exists for a full-time instructor in ophthalmology in the University of Miami School of Medicine, Miami, Florida. Right to limited amount of private practice included if individual has or obtains Florida license. Position is intended to be permanent, subject to the usual annual reviews, and unlimited potentialities for the future. Applicants should be academically minded, interested in teaching and research as well as practical ophthalmology. Write to Chief, Division of Ophthalmology, Department of Surgery, University of Miami School of Medicine, Jackson Memorial Hospital, Miami 36, Florida.

Listings of positions available should be sent to W. L. Benedict, M.D., 100 First Avenue Building, Rochester, Minn., by the fifth of the month preceding publication. They should include (1) type of physician wanted (ophthalmologist or otolaryngologist; diplomate, resident, etc.); (2) type of position to be filled; and (3) whom to write for further information. Unless otherwise requested, listings will be published once only.

ATLAS OF OTOLARYNGIC PATHOLOGY

The Atlas of Otolaryngic Pathology, published under the sponsorship of The American Academy of Ophthalmology and Otolaryngology, The American Registry of Pathology and The Armed Forces Institute of Pathology has been available to otolaryngologists and pathologists for the past year. This most excellent Atlas, prepared by Col. J. E. Ash (Ret.), M.D., and Muriel Raum, M.D., includes an outstanding group of presentations which will be of interest to clinicians as well as to persons working in otolaryngic pathology. The material was obtained largely from the files of The American Registry of Pathology. This unique and original series of photomicrographs, drawings and photographs of gross specimens is unparalleled.

The Atlas is obtainable from The American Registry of Pathology, 6825 16th St. N.W., Washington 25, D. C.

PAUL H. HOLINGER, M.D.

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